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AMERICAN JOURNAL OF OPHTHALMOLOGY

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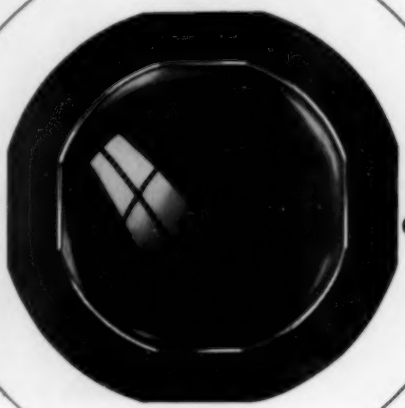

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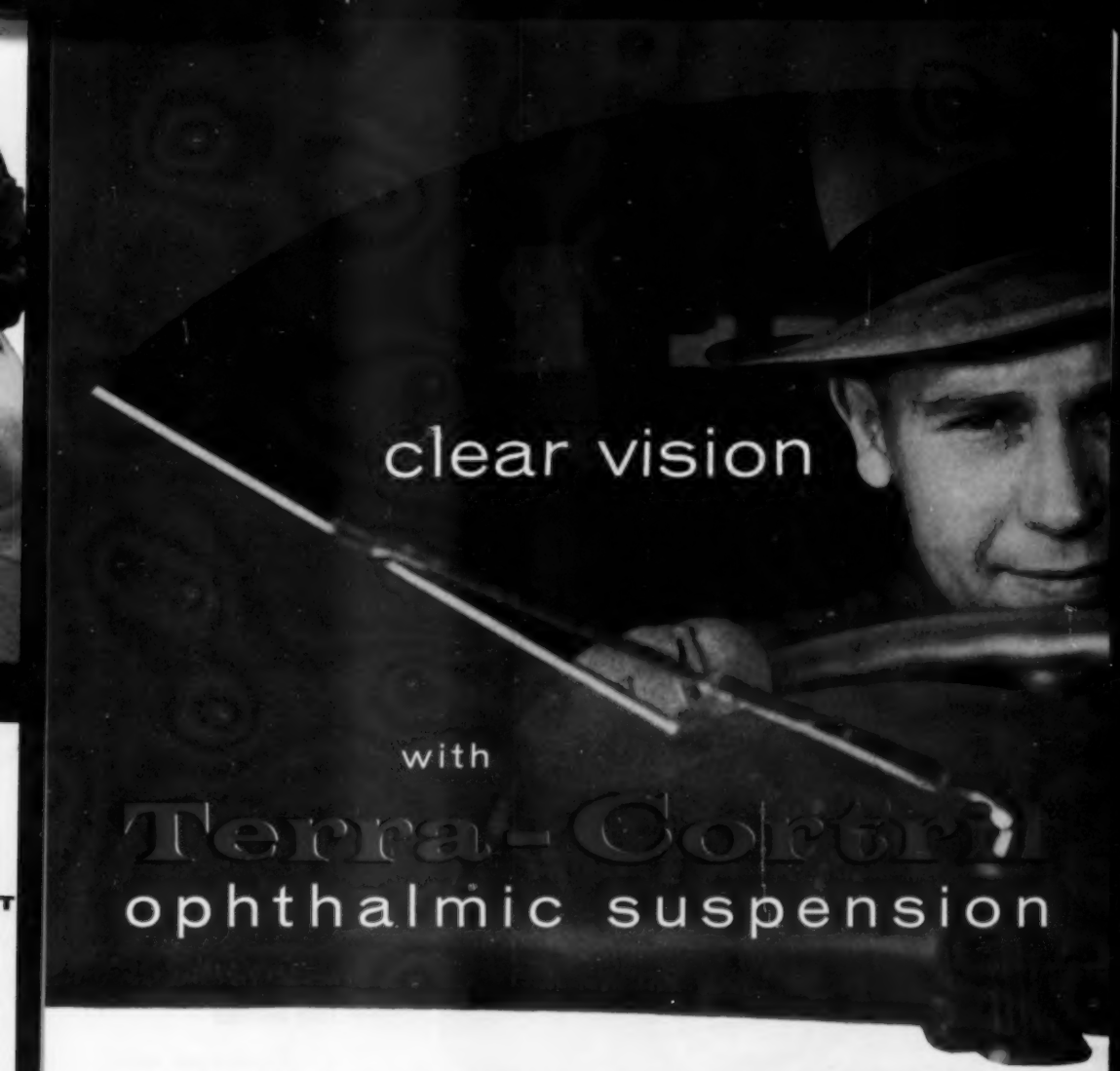

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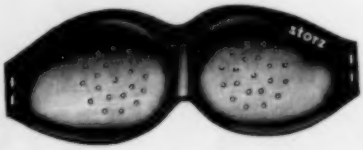

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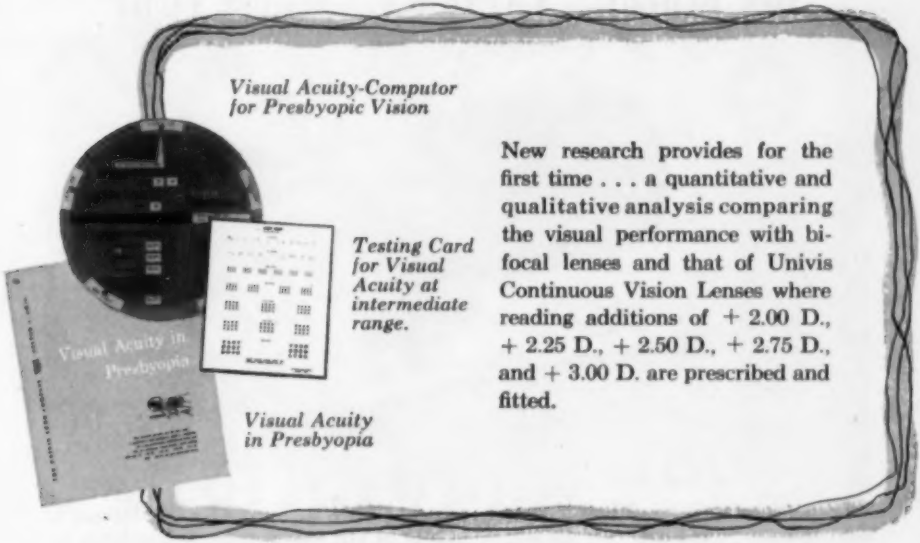
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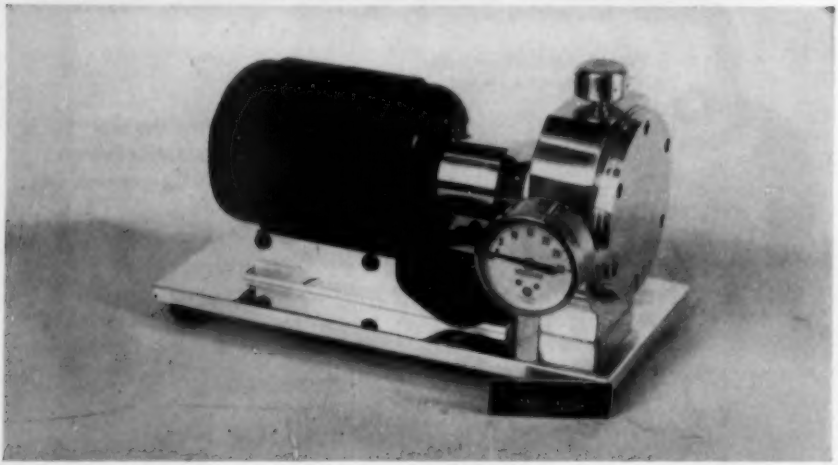

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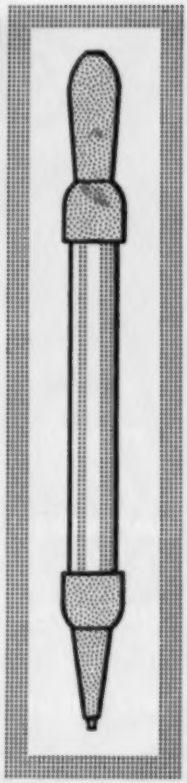

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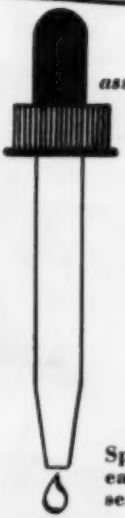

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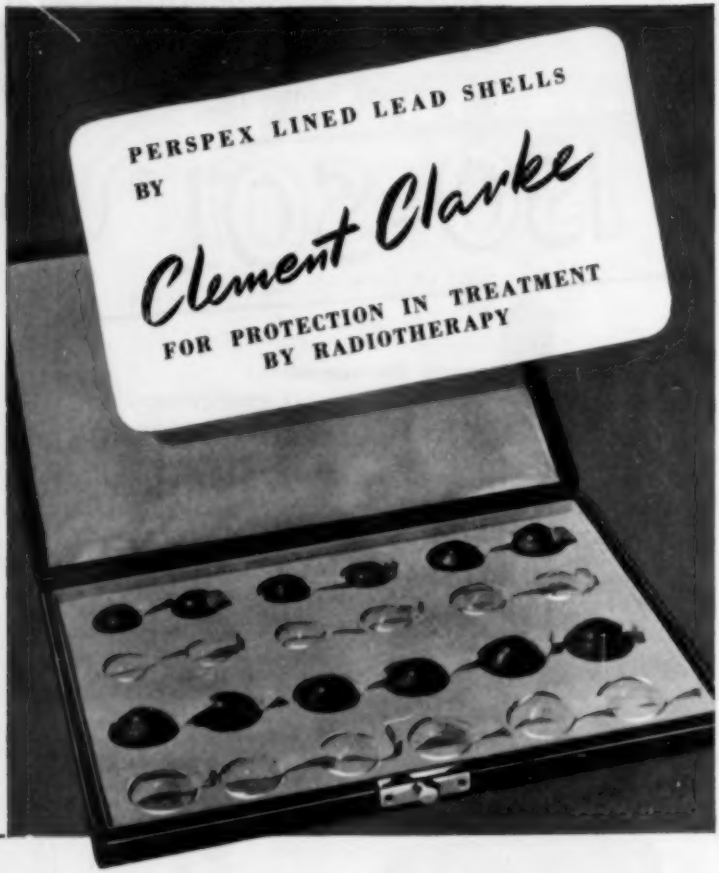

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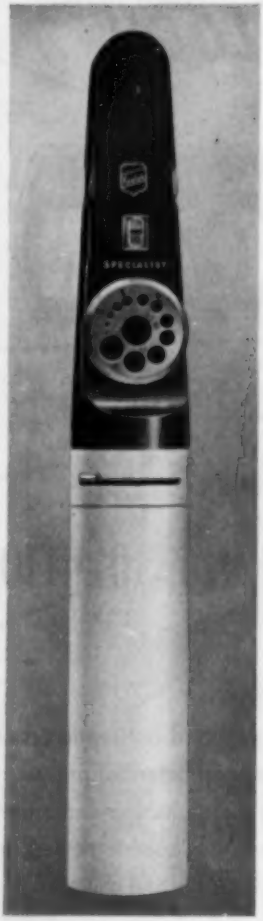

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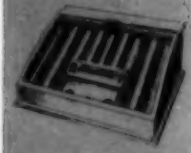

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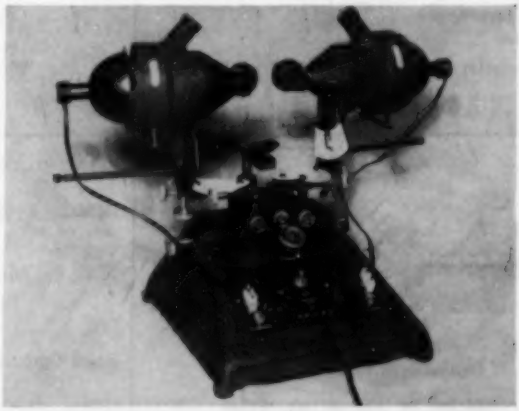

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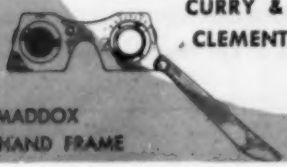

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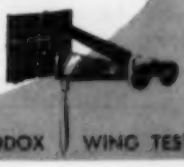

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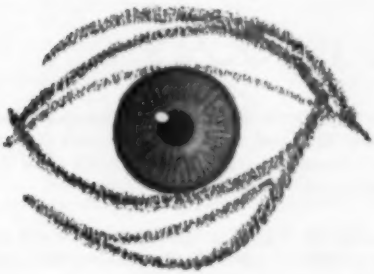

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1. Gettes, B. C., and Leopold, I. H.: A. M. A. Arch. of Ophth. 49:24 (Jan.) 1953.
2. Stolzar, I. H.: Am. J. Ophth. 36:110 (Jan.) 1953.

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Mr. B. was given 8 mm. intermediate segments on the following prescription: Right $+0.50$ $+0.75 \times 90$; Left $+0.75$ cylinder $\times$ 90, with a $+3.00$ reading add and a $+1.50$ intermediate add. The results were not satisfactory until we made a new pair of trifocals with 6 mm. segments. This pair did the trick nicely.

The conclusion arrived at from these and other cases is that when the reading addition is $+2.75$ or $+3.00$, or when the patient has poor accommodation, use the 6 mm. segment. Using the narrower segment gives a larger reading field and puts it higher in relation to the eye.

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AMERICAN JOURNAL OF OPHTHALMOLOGY

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ABSTRACTS

Vegetative physiology, biochemistry, pharmacology, toxicology; Physiologic optics, refraction, color vision; Diagnosis and therapy; Ocular motility; Conjunctiva, cornea, sclera; Uvea, sympathetic disease, aqueous; Glaucoma and ocular tension; Crystalline lens; Retina and vitreous; Optic nerve and chiasm; Neuro-ophthalmology; Eyeball, orbit, sinuses; Eyelids, lacrimal apparatus; Tumors; Systemic disease and parasites; Congenital deformities, heredity; Hygiene, sociology, education, and and history 583

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VOLUME 38 OCTOBER, 1954 NUMBER 4

DELAYED RESTORATION OF THE ANTERIOR CHAMBER*

THE EIGHTH PROCTOR LECTURE

PETER C. KRONFELD, M.D.
Chicago, Illinois

I sincerely appreciate the honor you have bestowed upon me. When the history of ophthalmology during the 20th century will be written, Dr. Francis I. Procter may come to be recognized as one of the greatest benefactors ophthalmology ever had.

For a long, long time the depth of the anterior chamber has served the ophthalmologist as an indicator of how the healing process after globe-opening operations or injuries is progressing. During the last 15 years we have witnessed a wave of renewed interest in the matter of the chamber depth after operations. The advent of gonioscopy as a method of clinical examination is at least partly responsible for this wave of new or renewed interest. The actual, in vivo, observation of peripheral anterior synechias after prolonged absence of the anterior chamber has made many ophthalmologists acutely conscious of the dangers of such absence.

Another reason for the renewed interest has been the demonstration, principally by Paul Chandler and associates,[1] of the pupillary block mechanism as a cause of shallowness of the anterior chamber after globe-opening operations. This form of delayed restoration of the anterior chamber has proved mechanically different from the more common form related, principally, to delayed healing of the operative wound. The treatment of the two forms is altogether different; the distinction between them is therefore of great importance and a new challenge to the ophthalmologist.

The principal purpose of this study was to determine the effects upon the ocular tension-regulating mechanism of delayed restoration of the anterior chamber by itself, uncomplicated by other factors, in the several distinct clinical situations in which such delayed restoration occurs.

If significant effects could be established, it was also the purpose of this study to weigh the risks entailed in delayed chamber restoration against the risks entailed in the procedures used for its treatment.

Thus there arises now the question: When may one speak of delayed chamber restoration? Answers may be obtained from experimental studies and clinical observations.

EXPERIMENTAL DATA

I have had the opportunity of studying the process of restoration of the anterior chamber on near-normal human eyes under the simple and well-controlled conditions of puncture of the anterior chamber with a hypodermic needle.[2, 3] By piercing the cornea at a small angle a long intracorneal track was obtained which nearly always became watertight immediately after withdrawal of the needle. In order to avoid injury to the lens, the tip of the needle, during its entire stay in the anterior chamber, was kept in front of the iris hugging the posterior surface of the cornea. In well over 500 anterior chamber punctures which my associates and I performed during the last 25 years, injury to the lens occurred only three times and was

* Presented at the University of California School of Medicine, San Francisco, December 4, 1953.

due to a faulty technique. The school of Amsler[3] has repeatedly testified to the innocuousness of the anterior-chamber puncture.

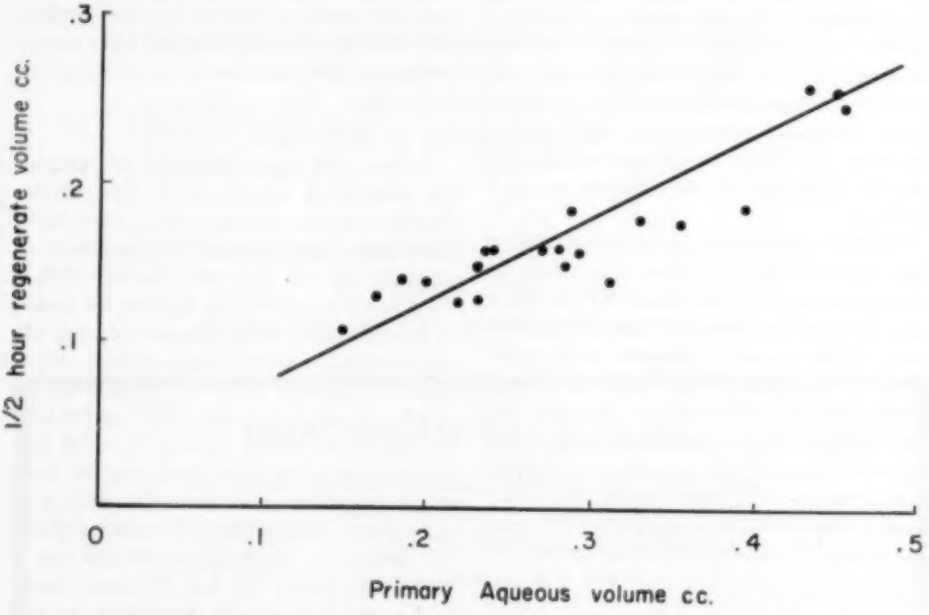
The near-normal eyes on which most of the work was done were those of Chinese patients, young or middle-aged, in fair general condition except for mild chronic dietary deficiencies concerning primarily proteins and vitamins of the B group. The eye condition was that of a mild, active or healed, retrobulbar neuritis due to the dietary deficiency. This Chinese form of optic-nerve disease related to dietary deficiency was very similar to the disease to become known later under the name of "camp-eyes." Included in the group of near-normal eyes were also a few cases of healed trachoma and a few heterotropias.

The rate of reformation of aqueous was studied on these near-normal human eyes by aspirating the contents of the anterior chamber twice, at time intervals varying from 10 to 90 minutes. The ocular tension was determined at intervals from 20 to 30 minutes.

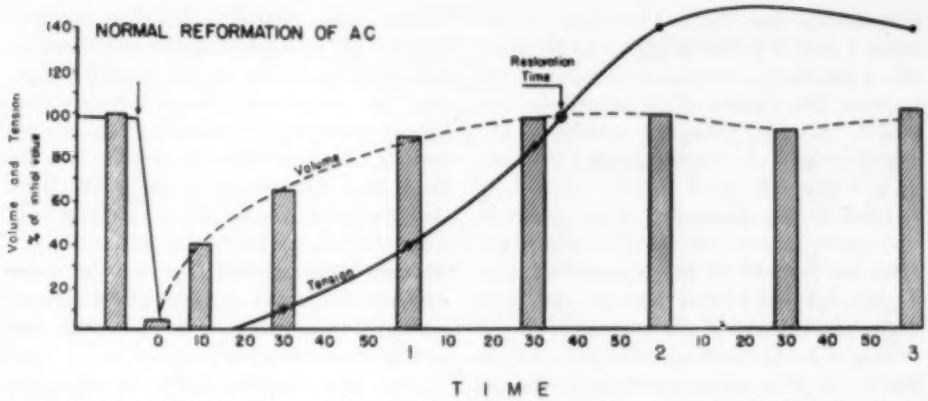
Thus a series of anterior chamber volume measurements just before and again, 10, 30, 60 or 90 minutes after the anterior chamber puncture was obtained. A series of volume measurements of the one-half hour regenerate, shown in Graph 1, is suggestive of a direct linear relationship between the original chamber volume and that of the half-hour regenerate.

The aspiration of greater original chamber volumes (V_1) appeared to cause more rapid reformation of aqueous so that the ratio regenerated volume (V_2) to initial volume (V_1) remains fairly constant. The absolute rate of regeneration of aqueous was greatest during the first 10 minutes (about 10 cu. mm. per minute) and then gradually decreased to an average rate of three cu. mm. per minute.

The dependence of the rate of reformation of aqueous upon the initial chamber volume became less pronounced during the second half hour after the initial puncture



Graph 1 (Kronfeld). A series of volume measurements of the one-half hour regenerate.



Graph 2 (Kronfeld). A comparison between the rate of reformation of the anterior chamber and the rate of restoration of the ocular tension.

so that at the end of the first hour the initially shallow chambers were completely reformed, the initially deeper chambers not completely reformed. The rate of fluid regeneration during the second half hour was approximately 2.08 cu. mm. per minute and not definitely dependent upon the initial chamber volume. Complete restoration of the chamber volume occurred, therefore, somewhat later in initially deep chambers than in initially shallow chambers.

A comparison between the rate of reformation of the anterior chamber and the rate of restoration of the ocular tension proved interesting (graph 2). During the first half hour after the puncture, the tension barely rose to a tonometrically measurable value while the fluid volume in the anterior chamber rose to 65 percent of the original value. During the second half hour, the rise in tension became very much faster while the rate of additional aqueous formation became markedly reduced over the rate prevailing during the first half hour. For the whole first hour the relation between volume and tension approximated the relationship determined experimentally by a number of investigators and formulated as $\log P = K_1 V + K_2$ by Friedenwald.[4]

In a special series of cases the second anterior-chamber puncture was performed at a time when the ocular tension had returned approximately to the original level. The data thus obtained showed that at the time at which the tension was restored to its original level the anterior chamber had also been restored to its original depth. The restoration time for the chamber fluid proved thus to be approximately the same or slightly shorter than the restoration time for the ocular tension so that for practical clinical purposes one may be substituted for the other.

Since the opportunity for determining the restoration time of the ocular tension presented itself in almost every instance of anterior-chamber puncture, considerable data accumulated over the years (table 1), showing a definite dependence upon the initial chamber volume.

For eyes of average chamber volume (0.25 cc.) the restoration time lies between 90 and 104 minutes. For eyes with unusually great chamber depth the average restoration

TABLE 1

THE AVERAGE RESTORATION TIME IN MINUTES

	Original Chamber Volume (cc.)			
	0.150–0.199	0.200–0.249	0.250–0.299	>0.299
Controls without Cataract	72	90	104	120
Controls with Cataract	83	91	97	115

time is about two hours. That figure is significant since it probably applies to the process of chamber restoration after cataract extraction. The volume of the crystalline lens (0.238 cu. mm.) plus the volume of the chamber that has to be regenerated may add up to 0.45 to 0.50 cc.

Table 1 also shows that with regard to restoration time of the chamber alone (not after the removal of the cataract) there is no significant difference between eyes with senile cataract and noncataractous eyes.

It may be of academic interest to record briefly the phenomena that occur during the period following the restoration time. The ocular tension goes through the well-known hypertensive phase.[5] The chamber volume becomes slightly but measurably reduced, probably as the result of an increase in the volume of the anterior or entire uvea. This reduction in chamber depth can be detected by photographic or direct measurement of the axial depth of the anterior chamber with Stenström's apparatus,[6] but usually remains unnoticed by the clinician.

Whether the phenomenon of the hypertensive phase in any way disturbs the healing of the cataract wound is difficult to say. There the data on human beings cease to give definite information.

From cataract extractions in dogs, I have learned that a smooth corneal, limbic, or slightly scleral incision securely closed with two to three appositional sutures, plus the natural resources, that is fibrinous exudation, remains water-tight in the vast majority of the entire postoperative course of such experiments. Typical aphakic chamber depth is reached after three to four hours and retained thereafter.

In a number of these operations it was possible to determine the ocular tension three to four hours after surgery, the result being consistently higher than the initial normal tension for the particular animal. There the hypertensive phase does not visibly disturb the reformation of the anterior chamber or the healing of the wound.

Very rarely, that is in less than five percent of all anterior chamber punctures in man done according to the standard technique, the needle track did not become water-tight immediately and remained fistulous for periods of a few hours to several days. In these cases the patency of the needle track could be recognized by slitlamp examination, a spindle-shaped space with optically empty contents being traceable through the entire cornea. The ocular tension of these eyes remained measurably or immeasurably low and in general very erratic.

Some characteristics of the fistulating eye, particularly its refraction, could be measured quite accurately. No gross bullous choroidal detachment was ever noted, but a slight change in the color of the fundus periphery nearly always occurred. The condition of fistulation after needle puncture was always self-limited.

Delayed restoration of the anterior chamber after cataract extraction in man

The observations after simple anterior-chamber punctures would lead one to expect that within the first three to four hours after the cataract operation the anterior chamber assumes full aphakic depth and retains this depth throughout the postoperative period. This expectation is borne out by clinical observations. Scheie[7] who routinely dresses his cataract cases eight to 10 hours after the operation finds most chambers to have reached aphakic depth at that time.

Twenty-four hours after surgery, at which time most surgeons inspect their cases, full aphakic chamber depth is the common finding. It occurred in 94 percent of a series of 749 cases of uncomplicated senile cataracts treated and followed for at least three months postoperatively at the Illinois Eye and Ear Infirmary.* These 749 cases were uncomplicated as far as the preoperative find-

* For these and other data I am indebted to Dr. Maurice D. Pearlman of the Department of Ophthalmology of the University of Illinois.

TABLE 2

INCIDENCE OF DELAYED ANTERIOR-CHAMBER RESTORATION AND SUBSEQUENT GLAUCOMA
749 UNCOMPLICATED CASES OF SENILE CATARACT

	Length of Delay (days)					
	1–2	3–4	5–6	7–8	9–12	Total
Number of cases of delayed restoration	31	21	14	9	8	83
Number of cases of glaucoma	0	0	0	1	5	6

ings and the operation were concerned. Corneoscleral sutures, principally of the McLean type, were used in practically all cases. Most of the extractions were done by the intracapsular method.

The ophthalmologist judges the progress of the healing of the cataract wound not only by the speed with which the anterior chamber assumes aphakic depth, but also on the basis of how this depth is maintained throughout the postoperative period. In our series the incidence of full aphakic chamber depth being maintained, as far as could be judged from daily examinations, throughout the entire postoperative period, was 89 percent (table 2).

Absence or near-absence of the chamber during some part of the postoperative period (except the first three to four hours) occurred with an over-all frequency of 11 percent, short episodes of absence being considerably more frequent than longer ones.

As criterion of abnormal chamber depth I have chosen the state described in the records of the Illinois Eye and Ear Infirmary as either complete absence or marked shallowness that is near-absence of the anterior chamber. I am well aware of the fact that a slightly shallow and even a moderately deep anterior chamber is abnormal after cataract operation. I have found, however, that individual variations in power of observation are more likely to cause differences in "findings" if the chamber is slightly shallow or moderately deep than if the chamber is absent or near-absent. By using the latter criterion throughout I am reasonably certain of uniformity with regard to the actual observations.

Such absence or near absence of the chamber occurred in our series with the frequencies shown in Table 2. The time of the onset or first recognition varied from the first to the 20th postoperative day, with somewhat higher frequencies during the first 10 postoperative days, and a slight peak during the first two postoperative days.

I believe that, in relation to the process of healing of the wound and of assumption of the permanent aphakic shape of the eye, absence of the chamber has the same meaning irrespective of the time at which it occurs.

The onset of the absence of the chamber could be correlated with a definite traumatic incident in about 40 percent of the cases. In the remaining 60 percent, the absence of the chamber developed without any demonstrable incident. In these cases the change from full aphakic to nonexistent depth tended to be gradual, that is took two to three days.

It is unfortunate that the delicate state of the recently operated eye precludes the application of many methods that would be most helpful in the study of postoperative absence of the chamber.

Tonometry which was so very helpful in the study of the process of chamber restoration after simple anterior-chamber puncture can, unfortunately, be applied only to a very limited number of operated eyes. In eyes with very low tension the tonometers of the Schiøtz type produce a very gross distortion of the entire cornea which may seriously disturb the state of coaptation of the wound lips. Souter's tonometer would probably be well suited for estimates of the ocular tension after cataract operation, if I

only had the necessary experience with the instrument which Dr. Verhoeff demonstrated to me 25 years ago.

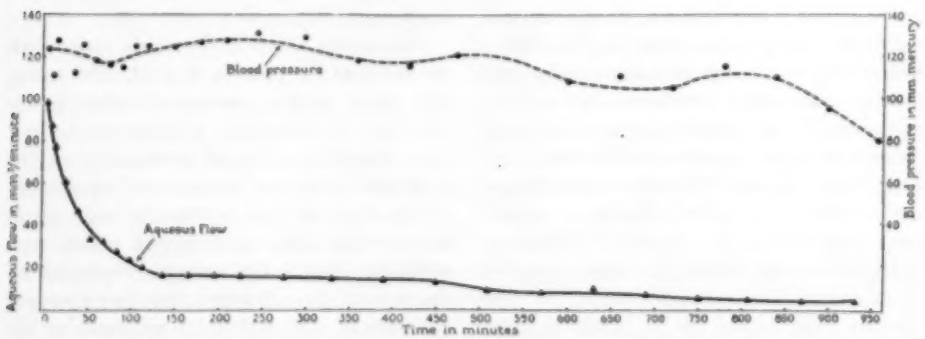
A safe, reliable method of tonometry applicable to the recently operated eye would be of great value in the study of normal and abnormal wound healing after cataract operations. Without such a method the healing process of the cataract incision has to be judged on the basis of biomicroscopic observations and Seidel's fluorescein test, in addition to estimates of the chamber depth and of the tactile tension. Despite the limitations of these methods the distinction between normal and abnormal healing processes can usually be made with a fair degree of definiteness.

Applying these methods to the eyes with absence of the chamber of our series one encountered, in about 30 percent of the cases, gross signs of leakiness of the wound, such as gaping of the incision between sutures visible through the flap, pale edema of the flap, a positive Seidel test from one of the suture sites or from a gap between the conjunctival wound lips, and a very low tactile tension. In another 20 percent of the cases, the same signs were present to a lesser or less definite degree. Surgical exploration of the wound in these cases almost invariably disclosed an unquestionable leak in the wound. Altogether an impressive amount of evidence indicative of leakiness of the wound as a cause of absence of the chamber was revealed by the study of the hospital records of our series.

This study also revealed that the absence of gross signs of fistulation does not rule out the possibility of fistulation, a conclusion which McGarry and I reached several years ago with regard to filtering operations.[8] This is again an instance where positive signs, such as visible dehiscence of the wound or a positive Seidel test, are conclusive evidence, but negative signs, that is, no visible dehiscence or a negative Seidel test, are inconclusive evidence.

The generally recognized causes of fistulization of a cataract incision are (1) incarceration of a tissue acting like a door jamb, (2) malposition of the wound lips caused by improperly placed sutures, (3) deep sutures acting as drainage wicks, (4) poor healing per se, and (5) accidental sclerectomies.

Leakiness of the wound can, naturally, account for absence of the chamber only if the capacity of the leak is greater than the capacity of the eye to regenerate intraocular fluid. The latter capacity, in the presence of a gross leak, was measured by Bellows[9] on dogs, and found to decrease rapidly (graph 3) with time. While there are no observations on human eyes that would correspond strictly to Bellows' experiments the latter suggest at least the possibility that after fistulation for some time the rate of fluid regeneration decreases to the point where it is smaller than the capacity of the leak. Thus



Graph 3 (Kronfeld). Results in Bellows' experiments on regeneration of the intraocular fluid.

a very small inconspicuous leak could account for persistent absence of the anterior chamber.

In the study of the mechanism of postoperative absence of the chamber the response of this condition to various therapeutic procedures has played an important part. The evaluation of these procedures is made difficult by the well-known fact that postoperative absence of the chamber is nearly always self-limited.

This is illustrated by 19 cases of ours in which the anterior chambers remained absent or near-absent despite various forms of conservative treatment. Finally, more or less in despair, the patients were discharged from the hospital with absent or near-absent chambers. In 17 of these 19 patients the anterior chamber was found to have reached aphakic depth at the time of the first visit to the outpatient department eight to 10 days later.

The form of treatment that is most logical in the face of absence of the chamber associated with an obvious gap in the incision is surgical exploration and repair by excision of incarcerated tissues and additional corneoscleral sutures. The surgeon usually has the opportunity of inspecting the wound thoroughly before and after the repair and to bring about good coaptation of all its external portion. After such repair the chances for restoration of the anterior chamber should be as good as after the cataract extraction and one would therefore expect full aphakic chamber depth to be reached in about 90 percent of the cases within the first 24 hours.

In an admittedly small series of 11 surgical repairs, prompt restoration of the anterior chamber occurred in six and very slow or no restoration in five cases. While there still remains the possibility of a minor wound leak not having been corrected by the surgical repair, the surgeons concerned with the case considered this possibility as very unlikely and were inclined to invoke factors not directly related to the wound as the cause of the delayed restoration.

In the presence of signs suggestive of distortion of the wound by one of the corneoscleral sutures or of wick action removal of the offending suture seemed to be called for and, in our series, was effective, in the sense of prompt restoration of the chamber, in about one out of eight cases. Cauterization of suture sites or parts of the conjunctival or corneoscleral wound suspected of fistulating was effective as chamber-restoring agent in about the same percentage of cases.

The only conservative measure that in cases of no apparent leak was effective in better than one out of nine trials was the topical application of strong cholinesterase inhibitors, such as eserine and di-isopropyl-fluorophosphate.

Applications of a strong mydriatic combination (epinephrine, atropine, and cocaine subconjunctivally) were tried in all cases where there seemed to be a possibility of a pupillary block. In a few of these cases a slight, but very transient deepening of the chamber followed the application of the mydriatic.

During the last few years, there has come to the fore the procedure of surgical restoration of the anterior chamber by means of air injections,[10-12] either alone or combined with release of the suprachoroidal fluid (Chandler[13]). The latter step, the release of the fluid between the detached choroid and sclera was first recommended by Verhoeff.[14] In our experience, an ophthalmoscopically visible choroidal detachment almost invariably accompanies postoperative absence of the chamber, gains in size, as the latter persists, and usually outlasts it by days or weeks. Our observations and interpretations with regard to choroidal detachment are entirely in accord with those of Hans Barkan expressed in his classical paper 38 years ago.[15]

The procedure of surgical inflation of the anterior chamber was used in our series in cases without gross, demonstrable leaks, usually after several of the conservative measures had failed.

The response to the surgical inflation of the anterior chamber with or without posterior sclerotomy varied within fairly wide limits. Three principal types of response could be distinguished:

1. The + response was characterized by the presence, 24 hours after the inflation, in the then very deep anterior chamber of a large amount of aqueous making up one half or more of the chamber contents. This amount increased steadily and the space occupied by air decreased at the same rate without any perceptible decrease in the total chamber depth. On the fifth postoperative day, or sooner, the entire chamber was filled with aqueous, with only traces of air bobbing around as the patient moved his eye.

2. The ± response was characterized, by a fair aqueous volume, corresponding to more than one half of full aphakic depth not later than the fifth postoperative day. This aqueous volume was reached slowly during the first five postoperative days, air being the main if not the only visible constituent during the first two days.

It was characteristic of both the + and the ± response that a steady gain was made during the first five days and that this gain was not lost or diminished after the fifth day.

3. The response to the surgical inflation was considered negative if no perceptible gain was made during the first five days. The amounts of aqueous in the chamber remained minimal and failed to show any trend to increase in any perceptible degree.

As basis for the evaluation of these observations may serve the rate of disappearance from the anterior chamber of air injected at the end of uncomplicated cataract operations.* That rate of disappearance is approximately the same as that under the conditions described here as the positive response to surgical inflation. One may therefore conclude that persistence of some air in the anterior chamber until the fifth postoperative day is not necessarily a sign of deficient aqueous regeneration.

TABLE 3

THE RESPONSE TO SURGICAL INFLATION OF THE ANTERIOR CHAMBER

	+	±	−
Inflation alone	6	7	6
Inflation plus posterior sclerotomy	12	6	2

Because of the well-established spontaneous "cures" of postoperative absence of the anterior chamber, I believe that the positive response should be considered an unquestionable success and the ± response a probable success of the surgical inflation. The results in our series are shown in Table 3.

The response to the combined procedure was on the whole better than that to the inflation alone. This difference becomes even more noticeable if one includes in the evaluation the seventh and eighth postoperative day during which both of the cases of negative responses to the combined procedure and two of the six cases of negative response to the inflation alone had gained full aphakic chamber depth.

Very obviously, the procedure of air injection into the non- or barely existent anterior chamber, with or without posterior sclerotomy, is infinitely more effective as a chamber-restoring procedure than any of the conservative measures mentioned in the foregoing. A possible mechanism of the procedure will be mentioned in the final chapter of this paper.

The effects of delayed chamber restoration upon the ocular tension-regulating mechanism were studied, principally, by random tonometric measurements, water-drinking tests if any reading above 25 mm. Hg (Schiøtz) was encountered, and gonioscopic examinations. It was found that delayed restoration by itself caused demonstrable disturbance of the tension-regulating mechanism only if it persisted for seven or more days. If the absence of the chamber lasted for nine or more days the incidence of

* For these data I am indebted to Dr. Fay Whitsell and Dr. M. Sternberg of Chicago.

TABLE 4

EFFECT OF DELAYED CHAMBER RESTORATION OF NINE OR MORE DAYS

	46 Cases
Incomplete follow-up	14
No glaucoma	11
Borderline	4
Glaucoma	17

secondary glaucoma became very considerable.

A few cases of secondary glaucoma occurred after chamber absences of less than seven days. A review of the records of these cases clearly showed that another complicating factor, usually in the form of uveitis, had been present. Such cases were carefully excluded from this study, just as were cases of secondary glaucoma after cataract extraction primarily due to uveitis or the sequelae of vitreous loss at the operation. Carefully excluded were also cases of early primary glaucoma aggravated by and therefore detected after the cataract operation. The condition of the other eye was most helpful in the detection of the latter group of cases.

Let us return now to the cases of "pure" glaucoma secondary to absence of the anterior chamber after cataract operation. In a group of 46 cases (tables 4 and 5) whose chamber had been absent for nine or more days we find a high incidence of definite glaucoma, but also a high percentage of eyes whose tension-regulating mechanism has not been demonstrably disturbed by the absence of the chamber. This phenomenon has been noted by previous writers, particularly by Chandler.[16] Between the frank glaucomas and the frank nonglaucomas one finds a small group of borderline cases without any demonstrable effect on the optic nerve over periods up to three years.

TABLE 5

GLAUCOMA SECONDARY TO DELAYED CHAMBER RESTORATION

	17 Cases
Controlled by miotics	8
Uncontrolled by miotics	4
Controlled by surgery	2
Uncontrolled by surgery	3

The glaucomas conformed closely to the general pattern described by Sugar,[17] Meyer and Sternberg,[18] Post and Harper,[19] and by us.[20, 21] Fortunately it often responds well and lastingly to miotics. It is now well established that the aphakic eye tolerates the strong cholinesterase inhibitors better and is much less disturbed by their side effects than the lens-containing eye. The progression of the optic-nerve disease is fairly slow so that even a state of seminormalization of the ocular tension by miotics may be adequate treatment in very elderly patients.

If miotics completely failed to lower the ocular tension, cyclodialyses were performed in our cases which, in another larger series of glaucomas secondary to delayed restoration of the anterior chamber, brought the tension down to the usual subnormal level in a little less than 50 percent of the cases.

The response of this form of glaucoma to cyclodialysis is not as good as that of chronic wide-angle glaucomas in which many surgeons achieve 70 to 80 percent successes, that is normalization of ocular tension. The peripheral anterior synechias which are so characteristic of the glaucomas secondary to absence of the chamber and which have to be broken in the area of the cyclodialysis may be the reason why in this form of glaucoma the results of the customary surgical treatment are not as good as in primary chronic glaucoma.

There arise now two questions, namely:

1. What factors determine whether eyes whose anterior chambers have been absent for nine or more days, become glaucomatous or not?

2. If they become glaucomatous what factors determine whether the glaucoma is controllable by miotics?

There is, I believe, a fairly definite answer to the second question. The gonioscopic find-

ings in eyes uncontrollable by miotics were invariably those of complete closure of the angle, that is solid peripheral anterior synechias to the anterior portion of the corneoscleral trabeculum or even more axial portions of the anterior angle wall. In eyes responsive to miotics, parts of the trabecular area were not covered by synechias and thus directly exposed to the aqueous, as far as one could judge from gonioscopic examination. A positive response to miotics was thus characteristically associated with at least partial accessibility of the trabeculum. The gonioscopic picture did not change in any appreciable way under the effect of the miotics.

Gonioscopic examination of all cases with delayed chamber restoration of nine or more days' duration disclosed peripheral anterior synechias far in excess of the amount seen after uncomplicated cataract extractions. Confirming earlier findings by Grossman and myself,[20] there was no apparent parallelism between the duration of the chamber absence beyond nine days and the extent of the synechias. In the glaucomatous eyes the extent of the synechias was, on the whole, greater than in the nonglaucomatous eyes, but there was much overlapping between the amounts of synechias in the two groups.

Gonioscopy thus proved useful in demonstrating the fact of delayed chamber restoration but did not, in our hands at least, serve to define the resultant outflow disturbance quantitatively.

No correlation could be found between the degree of pre-existent endothelial dystrophy (cornea guttata), of corneal striation, or of the postoperative state of irritation of the eye and the development or nondevelopment of glaucoma. As noted in a previous publication the structure of the anterior surface of the iris seemed to be a factor in the sense that irises with dense anterior border layers formed more solid synechias than irises with lacy, fluffy texture. Continued study, I believe, is likely to disclose one other, probably individual, factor governing the extent of synechias under the otherwise equal conditions prevailing during postoperative absence of anterior chamber.

Delayed restoration of the anterior chamber after glaucoma operations

Although delayed restoration of the anterior chamber is a fairly common phenomenon after certain glaucoma operations, representative and, at the same time, homogeneous series of cases are difficult to obtain from just one institution.

In close analogy to cataract extractions, two mechanically different types of delayed chamber restoration occur after glaucoma operation, one related principally to gross leakiness of the incision, the other caused most probably by a pupillary block mechanism.

The latter form has been dealt with in a recent study by Shaffer.[22] The former is a common phenomenon after filtering operations, particularly of the corneosclerectomy type, performed on cases of chronic, open-angle glaucoma.

Two series of 200 cases each of corneoscleral trephination and of iridencleises performed by the staff of the Illinois Eye and Ear Infirmary did not reveal any significant correlation between the result of the operation in terms of ocular tension one year after surgery and the length of the postoperative absence of the anterior chamber. In cases of corneoscleral trephination followed by absence of the chamber of 12 or more days' duration, the incidence of complications was significantly greater than in the cases of fairly prompt restoration of the anterior chamber. Some of these complications seemed to be related to massive incarcerations of uveal tissue in the trephine hole which were twice as frequent in cases of prolonged absence of the chamber as in cases of prompt chamber restoration.[23]

In a series of Lagrange sclerectomies of my own, I have witnessed the phenomenon of absence of the anterior chamber of eight or more days in approximately 20 percent of

the cases. During the last five years I have in every such instance used Chandler's air injection combined with release of the invariably present suprachoroidal fluid. The procedure has in every instance resulted in restoration of the anterior chamber, usually in accordance with the + response described in the foregoing.

The final outcome of the cases requiring surgical restoration of the anterior chamber was not significantly different from that of cases with prompt spontaneous chamber restoration.* On the whole, the effects of delayed postoperative chamber restoration were not very striking, probably because of the presence of other factors that exerted a strong influence upon the outcome of the surgical procedures.

A striking change for the worse occurred in narrow-angle glaucomas if a delay in chamber restoration followed an iridectomy. Of this sequence of events I collected seven cases including one from my own practice. In every one of these cases the iridectomy was performed between attacks which had not been severe and had responded fairly well to miotics. The iridectomy was recommended and performed to prevent further, more severe attacks. A delay in chamber restoration of three to four days converted the previously acute glaucoma into a chronic glaucoma requiring stronger and more frequent doses of miotics than prior to the iridectomy.

Again I am in complete agreement with Paul Chandler who in the Mark J. Schoenberg Memorial Lecture two years ago concludes his description of the technique of peripheral iridectomy in narrow-angle glaucoma with the following words:[24]

"The importance of tight closure of the wound with scleral sutures cannot be overemphasized. Persistence of a flat anterior chamber for even 24 hours, by promoting the formation of peripheral anterior synechias, may in some cases nullify the effect of the operation."

Tight suturing of the scleral wound after iridectomy has also been recommended by Haas and Scheie.[25]

The untoward effects of delayed chamber restoration after iridectomy are very striking and conspicuous, because of the very high percentage of successes, that is cures of the disease, obtained ordinarily with this operation.

Delayed restoration of the anterior chamber occurs, unfortunately, in a number of other situations of which I have not been able to collect large enough series to recognize any traits not exemplified by the situations already mentioned.

Conclusions and Summary

The observations recorded in the foregoing may be of value because of the number of cases upon which they are based. They support the view that delayed or disturbed healing of the operative wound is the principal cause of delayed restoration of the chamber. The frequency of this occurrence suggests the possibility of more subtle disturbances of the healing process in those cases of delayed chamber restoration in which the usual signs of a leaky wound are absent. No new observations pertaining to this specific and fairly common situation can be reported at this time except for the fact that surgical inflation of the anterior chamber combined with release of the suprachoroidal fluid has proven an eminently effective form of therapy.

The question now arises:

By what mode of action does the surgical inflation bring about its very remarkable results?

Since the air is retained in the anterior chamber in cases of obviously and intentionally gaping wounds, trephine canals, and sclerectomy openings, one may conclude that air can completely or partially block such passages just as it produced a pupillary block in Scheie and Frayer's experimental air in-

* In a recent personal communication Dr. P. A. Chandler has confirmed this observation.

jections into the anterior chamber.[26]

Close observation of Lagrange sclerectomies suggests a seal or valve action of the air bubble resting against the inner aperture of the wound. This very definite observation suggests a similar passage-blocking mode of action after cataract extraction. If that is the mechanism, there still is lacking an explanation of the fact—recorded repeatedly in this paper—that the rate of restoration of the anterior chamber varied within such wide limits after the assumed leak had been plugged.

As far as I can see, reduction in the amount of aqueous production could furnish an adequate explanation for the variations in the rate of restoration of the aqueous portion of the chamber. Such a reduction in aqueous production after prolonged fistulation is suggested by Bellows' experiments and has been considered as a cause of delayed chamber restoration on the basis of clinical observations (Byrnes,[27] Shaffer[22]).

An experimental approach to this possibility, with which we have been concerned for some time, has so far yielded only suggestive evidence. Reduction in aqueous formation, at this point, may be stated to be a possible and very likely factor in the still only partly understood mechanism of delayed chamber restoration.

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Since the reading of this paper, Dr. O. R. Tanner of Palo Alto (personal communication) and I have observed one case each of this type of acute hypertension due to pupillary block following air injection into the anterior chamber made to relieve postoperative absence of the chamber. Dilation of the pupil by strong mydriatics promptly relieved the hypertension.

References

1. Chandler, P. A., and Johnson, C. C.: A neglected cause of secondary glaucoma in eyes in which the lens is absent or subluxated. Arch. Ophth., **37**:740, 1947.
2. Kronfeld, P. C., and Lin, C. K.: Further studies on the regeneration of the aqueous in man. Proc. Soc. Exper. Biol. & Med., **39**:259, 1938; Tr. Am. Acad. Ophth., **42**:262, 1937.
3. Amsler, M., and Verrey, F.: De l'utilite pratique de la ponction de la chambre anterieure. Ophthalmologica, **105**:145, 1943.
4. Friedenwald, J. S.: Contribution to the theory and practice of tonometry. Am. J. Ophth., **20**:985, 1937.
5. Kronfeld, P. C.: The hypertensive response of the human eye to anterior chamber puncture. Am. J. Ophth., **36**:127, 1953.
6. Stenström, S.: An apparatus for the measurement of the depth of the anterior chamber, based on the principle of Lindstedt. Acta Ophth., **31**:265, 1953.
7. Scheie, H. G.: Personal communication.
8. Kronfeld, P. C., and McGarry, H. I.: The mode of action of iris inclusion operations. Tr. Am. Ophth. Soc., **48**:107, 1951.
9. Bellows, J. G., and Chinn, H.: Regeneration of the aqueous. Arch. Ophth., **24**:1144, 1940.
10. Selinger, E.: Injection of air into the anterior chamber after cataract extraction. Am. J. Ophth., **20**:827, 1937.
11. McMillan, J.: Injection of air as factor in maintaining filtration after corneoscleral trephining in glaucoma. Arch. Ophth., **22**:968, 1939.
12. McLean, J. M.: Gonioscopy in glaucoma operations. Tr. Am. Acad. Ophth., **45**:176, 1941; Arch. Ophth., **22**:968, 1939.
13. Chandler, P. A.: Primary glaucoma, complications of surgery. Tr. Am. Acad. Ophth., **53**:226, 1949.
14. Verhoeff, F. H.: Scleral puncture for expulsive sub-choroidal hemorrhage following sclerostomy-scleral puncture for postoperative separation of the choroid. Ophth. Record, **24**:55, 1915.
15. Barkan, H.: Postoperative detachment of the choroid. Proc. Sec. Ophth. AMA, 1915, p. 209; JAMA, **65**:1520, 1915.
16. Chandler, P. A.: Discussion of Meyer and Sternberg, Tr. Am. Acad. Ophth., **54**:339, 1950.
17. Sugar, H. S.: Concerning the chamber angle: Gonioscopy. Am. J. Ophth., **23**:853, 1940.
18. Meyer, S. J., and Sternberg, P.: Secondary glaucoma after cataract operation. Tr. Am. Acad. Ophth., **54**:326, 1950.
19. Post, L. T., and Harper, L. B.: A study of glaucoma secondary to cataract extraction. Am. J. Ophth., **36**:103, 1953.

20. Kronfeld, P. C., and Grossmann, E. E.: Relation of gonioscopic findings to incidence of secondary glaucoma in operative aphakia. Tr. Am. Acad. Ophth., **45**:184, 1941.
21. Kronfeld, P. C., and Haas, J. S.: Further study of the glaucoma due to peripheral anterior synechias following delayed restoration of the anterior chamber after cataract extraction. Tr. Am. Ophth. Soc., **42**:316, 1944.
22. Shaffer, R. N.: The role of vitreous detachment in aphakic and malignant glaucomas. Tr. Am. Acad. Ophth., in press.
23. Kronfeld, P. C.: The mechanism of filtering operations. Proc. Oto-Ophth. Soc. Pacific Coast, 1948, p. 330.
24. Chandler, P. A.: Narrow-angle glaucoma. Arch. Ophth., **47**:712, 1952.
25. Haas, J. S., and Scheie, H. G.: Peripheral iridectomy in narrow-angle glaucoma. Tr. Am. Acad. Ophth., **56**:589, 1952.
26. Scheie, H. G., and Frayer, W.: Ocular hypertension induced by air in the anterior chamber. Arch. Ophth., **44**:691, 1944.
27. Byrnes, V. A.: Treatment of delayed postoperative formation of the anterior chamber. Am. J. Ophth., **31**:1261, 1948.

CATARACT FORMATION FOLLOWING BETA-RAY RADIUM THERAPY*

Report of four cases

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One of the outstanding advances in ophthalmology during the past several years has been the development of beta-ray radium therapy. It has proved useful in such varied conditions as papillomas, corneal vascularization, and corneal scarring, as well as in the preoperative and postoperative management of keratoplasty. Our own experiences have corroborated those of others and have demonstrated that this is indeed a useful and valuable form of therapy.

Until quite recently we considered, as did many others, that beta radiation, when used with due precautions, is an innocuous method and that cataract formation does not follow its use. Unfortunately, we have learned that this generalization is not correct. The primary purpose of this communication is to put on record the case histories of four patients who have recently presented themselves with cataracts, in one instance bilateral, following beta irradiation delivered in varying dosages, chiefly with the beta-ray radium applicator of Iliff. The single patient who was also treated with the radium-D applicator was given such small dosages with it that, in the light of the other case histories and for the purpose of this paper, his treatment by this means can fairly be disregarded.

Our second purpose in this communication is to record our present concept of dosage, which has developed chiefly as a matter of clinical evolution, in which, of course, the appearance of cataracts in these four patients has played an important role.

These cases have been discussed informally at a number of medical meetings since the spring of 1952, but have not previously been recorded. We can find no record of similar cases in the periodic literature. Hughes, in an extended discussion of beta radiation therapy in the 1952 *Transactions* of the American Ophthalmological Society, mentioned cataract formation in two patients (three eyes) following the use of this technique for primary vascularizing keratitis. In the first case 44 gm. sec. was applied over an 18-week period, and in the second, 51 gm. sec. was applied over a 16-week period. Evidences of cataract formation appeared at 30 and 35 months, respectively, after the first treatment.

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TABLE 1

BETA IRRADIATION FOR PTERYGIA—CASE 1

Date	Dosage in Minutes O.D.	O.S.	Applicator
Aug. 8, 1947	10		Iliff
Aug. 11, 1947		10	Iliff
Aug. 15, 1947		10	Iliff
Aug. 23, 1947		15	Iliff
Sept. 2, 1947	15		Iliff
March 10, 1948		3	Radium D
March 28, 1948		2	Radium D
April 11, 1948		2	Radium D
April 30, 1948		3	Iliff
May 30, 1948		2	Iliff
TOTAL	25	40	Iliff
		7	Radium D

Unfortunately, few descriptive details are supplied. We have occasionally observed cataract formation after primary vascularizing keratitis not treated by irradiation, and we have frequently observed an "amber-colored nuclear haze" after senile cataracts. As we shall point out, the cataracts which we are recording in no way resemble the ordinary senile type of cataract.

Whether or not these particular cataracts were similar to those which we are reporting, the important consideration, it seems to us, is that all ophthalmologists who feel, as we do, that beta radiation is a valuable addition to ophthalmic therapy should place on record, for the common good, their adverse as well as their favorable experiences with it.

CASE HISTORIES

CASE 1

A 29-year-old white man was first seen June 3, 1947, with bilateral malignant pterygia, more advanced on the left side. Vision was 20/20 in the right eye and 20/25 in the left. No intraocular abnormalities were evident when the pupils were dilated. On July 10th, a modified bilateral McReynolds transplantation operation was performed. On August 8th, a beginning bilateral recurrence was observed, slightly more marked on the left. Between that date and September 2nd, (table 1), the patient received treatment with the Iliff applicator for a total of 25 minutes over the right eye and for a total of 35 minutes over the left eye. Treatment was apparently effective.

On March 10, 1948, a slight recurrence was observed on the left side. Between that date and April 11th, the radium-D applicator was used for a total of seven minutes over this eye. There was no noticeable clinical improvement. Additional treatments were therefore given with the Iliff applicator April 30th and May 30th for a total of five minutes. When the patient was examined November 12, 1947, both pterygia appeared well controlled and, when the pupils were dilated, all intraocular structures appeared normal.

He was not seen again until October 12, 1951, when he presented himself complaining that for the past 48 hours vision on the left side had been blurred and that he felt as if something was in the left eye.

Vision was 20/30, correctible to 20/20, in each eye. Examination disclosed no foreign body and staining was negative. A moderate conjunctivitis was present. The pterygia had not recurred, but a number of telangiectatic vessels were observed in both eyes around the areas which had been irradiated. When the pupils were dilated, cataractous changes were noted in the nasal periphery of each lens, directly under the areas which had been irradiated, and more advanced on the left side than on the right.

Slitlamp study showed subcapsular changes anteriorly and posteriorly but more developed equatorially. Numerous vacuoles in the affected areas gave them a golden, cobblestone appearance. In the more advanced lesion on the left side the process extended almost to the posterior pole. Otherwise, the processes in both eyes appeared identical under the slitlamp.

CASE 2

A 39-year-old white woman was first seen June 20, 1947, with the complaint that her eyes became tired and red after reading. Vision was 20/20 in both eyes. There was an early malignant pterygium in the right eye,

TABLE 2

BETA IRRADIATION FOR PTERYGIA—CASE 2

Date	Dosage in Minutes O.D.	O.S.	Applicator
Aug. 8, 1947		10	Iliff
Feb. 7, 1948	2		Iliff
March 28, 1948		10	Iliff
April 1, 1948		10	Iliff
May 3, 1948	10		Iliff
TOTAL	12	30	Iliff

and a similar, more advanced lesion in the left eye. When the pupils were dilated, all intraocular structures appeared normal.

A modified McReynolds operation was performed on the left eye July 14th. Late in July, recurrence of the pterygium was observed and the Iliff applicator was used for 10 minutes August 8th (table 2). It was used again over the same eye March 28 and April 1, 1948, for a total of 20 minutes. The lesion in the right eye was treated with the Iliff applicator February 7 and again May 3, 1948, for a total of 12 minutes.

When the patient was next seen, January 21, 1952, she complained of blurring of vision. Vision was 20/25 in the right eye and 20/30 in the left, correctible to 20/20 in both eyes. The pterygia appeared well controlled in both eyes, but the areas which had been irradiated were blanched and were surrounded by telangiectatic vessels. When the pupils were dilated, the intraocular structures on the right side appeared normal. On the left side, directly beneath the irradiated area, cataractous changes were observed in the nasal area of the lens, in the form of an anterior subcapsular vacuolization with some posterior subcapsular extension. As in Case 1, the vacuoles present gave the affected area a cobblestone appearance.

This patient was re-examined June 15, 1953, with the same findings as on the last examination almost 18 months earlier.

CASE 3

A 43-year-old white man was seen January 31, 1948, complaining of red, irritated eyes and recurrent styes. Vision was 20/20 in the left eye and 20/25 in the right, correctible to 20/20. Multiple chalazia were present in the lids on both sides. A vascular pterygium was present on the right side, in the nasal area. When the pupils were dilated, all intraocular structures appeared normal.

The chalazia responded promptly to standard medical and surgical treatment. A modified McReynolds transplantation operation was performed February 19, 1948, for the pterygium in the right eye. On March 3rd, when a regrowth of vessels and an early recurrence of the pterygia were observed on slitlamp examination, the Iliff applicator was used for five minutes over the surgical field (table 3). Additional treatments were given between March 10th and June 12th for a total dosage of 14 minutes.

When the patient was next seen, January 18, 1952, he complained of reading difficulties. Vision in the right eye was 20/30 and in the left 20/25, correctible to 20/20 and J1 on each side. There was no recurrence of the pterygium, but telangiectatic vessels were observed around the area irradiated in the right eye.

Slitlamp examination, with the pupil dilated, revealed cataractous changes in the nasal periphery of the right lens, directly under the irradiated area. Subcapsular vacuolization was present both anteriorly and posteriorly but was more marked equatorially. The affected area, as in the preceding

TABLE 3

BETA IRRADIATION FOR PTERYGIUM—CASE 3

Date	Dosage in Minutes O.D.	Applicator
March 3, 1948	5	Iliff
March 10, 1948	5	Iliff
April 8, 1948	3	Iliff
May 12, 1948	3	Iliff
June 12, 1948	3	Iliff
TOTAL	19	Iliff

case, had a light golden hue. The left lens was normal.

CASE 4

A 35-year-old white man was first seen March 11, 1949, with a history of two previous operations for a pterygium in the right eye. He had a large recurrence at this time. On March 12th, the standard operation for pterygium was performed. Forty-eight hours later he received five minutes of beta radiation with the Iliff applicator. This dosage was repeated March 16th and again March 18th.

On April 19, 1950, a small recurrence (pseudopterygium) was observed. It was excised and five minutes of beta irradiation were delivered with the Iliff applicator immediately after operation. This dosage was repeated April 20th and again April 21st.

The patient was seen at irregular intervals between the latter date and February 8, 1953, always with negative findings. On February 8th, for the first time, anterior and posterior capsular changes were observed in the region of the equator, exactly the same in character as those observed in the four eyes already described. Telangiectatic vessels were also present. Visual acuity was 20/20 with correction. When the patient was next seen, May 12, 1954, there had been practically no changes in the findings recorded 15 months earlier.

COMMENT

Our own feeling has always been that the large proportion of recurrences after operations for pterygia justify any reasonable attempt to control the superficial vascularization and fibrous overgrowth associated with the reappearance of the lesions.[2] We have used beta irradiation, practically always with the Iliff applicator, for this purpose in vascular and malignant pterygia from the time the method was first introduced, with a gratifying proportion of successes. We have, however, reduced the dosage materially and changed the technique of application as time has passed.

The dosage employed in the 1947 to 1949 period, when these four patients were treated, was delivered entirely in three cases, and chiefly in the fourth case, with the Iliff applicator according to Iliff's[3] basic recommendations in 1947:

> Ten minute applications, delivering doses of four gram seconds, can be repeated after an interval of one week, and again the second week, the total dose remaining well below the contact skin erythema dose of 18 gram seconds. Such a course of therapy could be repeated after an observation period of two to three weeks and at intervals as found necessary for the patient's improvement.

During the 1947 to 1949 period, in which the cases just described were treated, the indication for both the original application and for repeated irradiation was the appearance of the recurrent pterygium clinically and under the slitlamp. The second application was made within two weeks of the first because a cumulative effect was desired. The third application, however, was not made for two weeks or more after the second because it was now desired that the cumulative effect, if any, should be minimal. All applications were made with discretion, but no particular maximum was set up.

In the four cases (five eyes) upon which this report is based, the total dosage with the Iliff applicator was variously 25 minutes, 40 minutes, 30 minutes, 19 minutes, and 30 minutes. Cataract formation did not occur in the second eye in Case 2, in which this method was used for 12 minutes.

In 1948, in an endeavor to determine the practicability of treating glaucoma by obliteration of the vascular supply of the ciliary body, one of us (G. M. H.[4]) conducted a series of experiments in which beta irradiation was applied to the ciliary body in rabbits; the applications were for varying amounts and over varying periods of time. None of the animals presented clinical evidence of cataracts but well-developed microscopic changes were found in the lens substance of animals exposed to 120 minutes of irradiation and killed 42 days later. Similar but less well-developed changes were also

found in animals killed nine days after only 30 minutes of irradiation had been applied.

In the light of these clinical and experimental observations, we altered both our technique of application and our dosages of beta-ray irradiation after surgery for pterygia. The routine which we are now employing, and which has gradually evolved over the last four and one-half years, is as follows:

Beta irradiation delivered by the Iliff applicator is now used after certain operations for pterygia as soon as the necessary surgery has been completed. The dosage does not exceed five minutes unless the denuded area is unusually large. Then it may be necessary to make two applications, for a total of 10 minutes, to be certain that all areas have been treated. Care is taken that the applications are not overlapping. No further treatment is given at this time.

If at the end of six to eight weeks, a recurrence is observed (usually a pseudopterygium), it is at once excised and another five-minute application is made. We have found that the decreased exposure time seems to produce results entirely comparable to those produced by the longer exposures employed in 1947 to 1949, and the reduction of dosage, combined with the altered technique, theoretically at least, should eliminate the risk of cataract formation. It is still too soon to speak categorically, but we have not observed this complication in any patient treated since our dosages were reduced.

We believe that our present technique, which involves the application of beta irradiation immediately after the operation, is the reason why the smaller dosages are as successful as the large dosages formerly used. Formerly, our policy was to delay application until new vessels had begun to form or old ones were evident. Our present policy is more logical, in the light of the known principle of irradiation therapy that the newer the vessel, the more readily it responds to radiation.

It is unfortunate that two of these four patients, although requested to return for further examination, failed to do so. The other two, however, have been observed, respectively, for 15 months and 18 months since their cataracts were first discovered. It is interesting and reassuring to find that, over this period, the changes originally observed have shown almost no progression.

We are quite aware of the possibility that, by the law of averages, some patients subjected to beta irradiation would eventually develop cataracts, regardless of therapy. It should be stressed, however, that the lesions described in these five eyes are not senile cataracts.

In all five eyes changes were confined to the areas which had been irradiated. No other areas were affected. In all five eyes the processes were remarkably similar. The peripheral subcapsular areas were chiefly affected. Vacuolization did not seem to extend along the lens fibers into the cortex, even in the equatorial region. Posterior subcapsular changes seemed somewhat more extensive than anterior changes, though this observation may perhaps be partly explained by the inaccessibility of the peripheral anterior subcapsular area to complete examination by the slitlamp. Finally, telangiectatic vessels were observed about the irradiated areas in all five eyes.

As our experience has increased, we have come to feel that blanching in the irradiated area, if it lasts longer than three days, may indicate overdosage. That impression has been borne out by the appearance of telangiectatic vessels in some of the irradiated cases in which this phenomenon was observed. In addition to our observations in these five eyes, we have demonstrated such vessels in a number of other patients treated with beta radiation. They are now being carefully followed for possible future cataract development.

Summary

Four cases (five eyes) are reported in which cataracts followed the use of beta radiation therapy for the control of recur-

rent pterygia. The total dosages, which varied from 19 to 40 minutes, were delivered by the Iliff applicator in 1947 to 1949 by the technique originally advised by Iliff.

In the technique now employed in the management of pterygia, a single application, which does not exceed five minutes in any area, is made routinely immediately after surgery, when new vessels are most responsive to radiation. The application is not repeated unless there is a recurrence (pseudopterygium). When this happens, secondary surgery is followed immediately by a five-minute application with the Iliff applicator.

Follow-up observations in two of the four cases reported in this paper showed practically no change in the eyes after periods of 15 and 18 months.

It is hoped that other observers will put on record their experiences with cataract formation and other complications following beta radiation. This is an extremely valuable method in various fields of ophthalmology and the pooling of experiences will help to eliminate any hazards which may be connected with its use.

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REFERENCES

1. Hughes, W. F., Jr.: Beta irradiation in ophthalmology. Tr. Am. Ophth. Soc., **50**:469, 1953.
2. Haik, G. M., Waugh, R. L., Jr., Lyda, W., and Ellis, G. S.: The management of pterygia with particular reference to recurrent pterygia. Southern M. J., **45**:832, 1952.
3. Iliff, C. E.: Beta-ray radium applicator for ocular use. Preliminary report. Arch. Ophth., **38**:827, 1947.
4. Haik, G. M., Breffeilh, L. A., and Barber, A.: Beta irradiation as a possible therapeutic agent in glaucoma: An experimental study with the report of a clinical case. Am. J. Ophth., **31**:945, 1948.

LATE INFANTILE AMAUROTIC IDIOCY*

A CLINICO-HISTOPATHOLOGIC STUDY

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Among the diseases with basic disturbances of the lipoid metabolism of a cell system the amaurotic idiocies take a special place because of the characteristic involvement of the ocular fundus.

Taking the group of primary lipoidoses in general, the enzymatic dysfunction of cells which becomes evident by accumulation of lipoids within these cells and their transformation into foam cells may remain confined to ectodermal cells, such as the ganglion cells of brain and retina, or may include mesodermal cells, causing widespread visceral involvement in addition to the cerebral and ocular manifestations.

It is not clear whether the intracellular accumulation of lipoid is due to an increased synthesis or a decreased splitting of certain lipoid compounds.

The physical and chemical properties of the stored lipoids vary in the different members of the primary lipoidoses. Thus, cholesterol and its esters prevail in the foam cells of the eosinophilic xanthomatous granuloma (Schüller-Christian-Hand's disease) and in its early and more accelerated generalized form (Letterer-Sieve's disease). Immense intracellular deposits of the cerebroside kerasin characterize Gaucher's disease. In dysostosis multiplex (Hurler's syndrome or gargoylism) more complex compounds of lipoids and protein fill the foam cells (Oxford Medicine[1]).

The group of amaurotic idiocies which comprises chiefly three disorders, namely, the

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infantile familial form (Tay-Sachs), the juvenile form (Stock-Vogt-Spielmeyer, Batten-Mayou), and the essential lipoid histiocytosis (Niemann-Pick's disease), has been recognized as predominantly a disturbance of phosphatide metabolism. In this connection, the importance of phosphoric acid derivates as hydrophil substances regulating the permeability of the cell membranes and thus the intermediary cellular metabolism has been stressed (Rintelen[2]). The stored lipoids in the foam cells of the juvenile form have been demonstrated as a mixture of phosphatides and cerebrosides (Givner and Roizin[3]).

In recent years the similarities rather than the dissimilarities between the various forms of amaurotic idiocy have been emphasized. Thus, Tay-Sachs and Niemann-Pick's disease, which are still considered by most as two different clinical entities, may, according to some investigators, be only variants of the large group of phosphatide lipoidoses, one showing the cerebroretinal symptom complex only, the other the hepatosplenic type only, or mixtures of the two (Sorsby[4]).

In this connection, the identical appearance of the fundus picture in Tay-Sachs and Niemann-Pick's disease, if the latter shows retinal involvement, bears emphasis besides the fact that both disorders occur almost exclusively in the Jewish race.

The similarities between the infantile and juvenile forms of amaurotic idiocy are less obvious, at least clinically, since the ophthalmoscopic picture of the latter is dominated by pigmentary disturbances.

The study of a group of intermediate cases with delayed onset of such symptoms as are usually associated with the infantile form, and with the additional finding of pigmentary degeneration, first suggested that the boundaries between the two are not as sharp as they were assumed to be (Bielschowsky[5]).

The pigmentary disturbance in the ocular fundus associated with the juvenile form may be either one of two varieties, namely, a diffuse degeneration resembling retinitis pigmentosa such as was described by Stock,[6] Vogt,[7] and Spielmeyer,[8] or one confined to the macular region as in the cases first described by Batten and Mayou[9] and appropriately termed cerebromacular degeneration.

In both varieties the visual disturbance, which may be the first manifestation of the disease, has its onset around the age of six years. Mental deterioration becomes noticeable about the same time or soon afterward. Epileptic seizures may develop a few years later and death occurs usually between the ages of 17 to 20 years. In the patients with diffuse pigmentary degeneration the disease appears to run a more accelerated course.

The juvenile and the late infantile and the intermediate forms do not occur as exclusively among the Jewish race as the infantile type.

The following case is of interest because ocular findings typical of the juvenile form of amaurotic idiocy with diffuse pigmentary degeneration were combined at an unusually early age with a symptom complex characteristic of the infantile form. Richter and Parmelee[10] who discussed in detail the general and neurologic findings in this case classified it as a late infantile amaurotic idiocy.

Report of case

G. D., a boy, aged five years, was admitted to the Presbyterian Hospital with following history:

He was the second child of apparently healthy non-Jewish parents, the father, aged 51 years, the mother, aged 32 years. The first child and two children of the patient's father from a previous marriage were normal in every respect. The paternal family history was negative; the maternal history revealed an uncle who was not normal and died at the age of one year. The nature of his abnormality and the cause of his death could not be ascertained.

The patient's birth was normal. The birth weight was nine pounds and the postnatal development was normal to the age of 14 months. He was breastfed for eight months.

He was able to hold his head up at the age of five months, sat up at eight months, and walked and said his first word "mama" at the age of one year.

At 14 months some unusual behavior was noted for the first time. Without preceding illness the child became fussy, lost his appetite, and stopped walking and sitting up.

During the following year both arms and legs became paralyzed and feeding became increasingly difficult because of gagging. He also had measles and chickenpox at the age of two or three years.

At the time of his admission to the hospital, the child was emaciated, weighing only 21 pounds. The salient features of the general examination at that time were:

1. *Physical examination* (Parmelee). The child showed no signs of any awareness of his surroundings and periodically uttered a weird cry. The circumference of the head was 18.75 inches. Chest and abdomen were normal. There was constant twitching of arms and legs, and all deep reflexes were markedly increased, but there was no Babinsky sign.

The pupils were dilated and there appeared to be a slight reaction to strong light.

2. *Neurologic examination* (Bassoe-Richter). The position of the extremities suggested decerebrate rigidity with hyperflexion of the elbows, wrists, metacarpophalangeal joints, and less pronounced in the joints of the lower extremities.

3. *Laboratory findings.* The urine was normal, blood and spinal Wassermann negative. The total protein content of the spinal fluid was 41.6 percent.

The child died nine months after his admission to the hospital from bronchopneumonia due to extreme aspiration of gastric contents.

Autopsy findings. Aside from the pulmonary findings there were no gross or microscopic abnormalities of the inner organs.

The brain weighed 416 gm. There was extreme atrophy of the hemispheres, brainstem, and cerebellum with external and internal hydrocephalus ex vacuo.

The cross sections of the optic nerves measured two mm. in diameter, and optic chiasm and tracts were correspondingly reduced in size.

Histologic examination of the central nervous system revealed two salient features, namely, generalized degeneration of the ganglion cells and diffuse tremendous, probably compensatory, proliferation of neuroglia.

A coexisting diffuse, fibrous leptomeningitis was interpreted as secondary to irritation by the end-products of degeneration pouring into the subarachnoidal space over a long period.

Ocular findings

Vision appeared to be nil during the entire time of observation. Lens and vitreous body were clear in both eyes. The fundi had identical appearance (fig. 1). The optic discs were grayish white. Their outlines were slightly fuzzy and no pattern of the cribriform plates could be made out.

The fundus in general had a mottled ap-

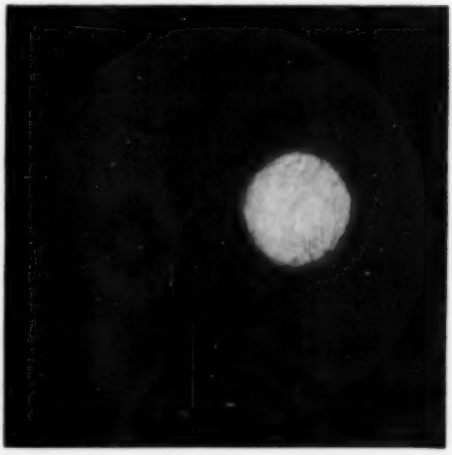

Fig. 1 (Klien). Ophthalmoscopic appearance of fundus of right eye: Grayish white color and fuzzy outline of optic disc, shadowy remnants of retinal vessels, mottled appearance of fundus, round, dark area in fovea centralis with punched-out appearance.

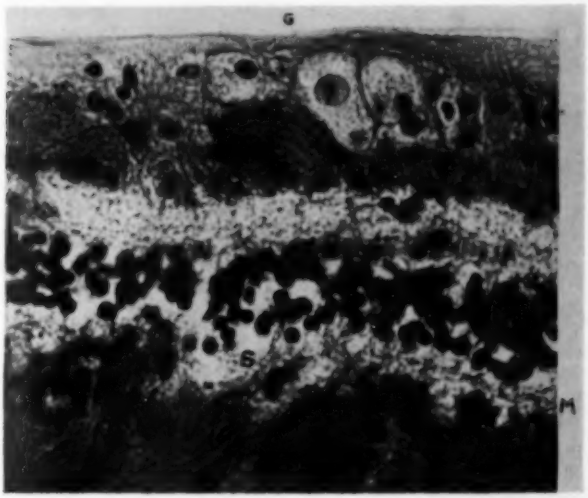

Fig. 2 (Klien). Swollen and disfigured ganglion cells (G) containing foamy granular substance (prelipoid), fatty granular cells (scavenger cells) in inner nuclear layer (S), massive glial plaque replacing outer retinal layers (M). (Hematoxylin-eosin.)

pearance with a few areas of intensified dustlike pigment accumulations. The macular reflexes were missing and the area of the fovea centralis appeared to be a round, slightly darker spot.

On close inspection traces of the retinal vessels could be made out. Only the larger branches were discernible, the arteries as faint grayish lines, the veins mainly as a thin upper and lower branch on the discs.

Histologic examination of the right eye. The globe was fixed in formalin, embedded in celloidin, and sectioned serially.

The anterior segment of the globe was normal. The pathologic findings in the posterior segment were limited to retina, optic nerve, and the posterior vitreous.

There were two main pathologic changes in the retina—a complete destruction of the first neuron with proliferation and migration of pigment epithelial cells, and swelling and degeneration of the ganglion cells.

The ganglion cells were reduced in number and the remaining cells revealed various degrees of degeneration. There was balloonlike swelling and disfigurement of many of them (fig. 2). Some had well-preserved nuclei which, however, were displaced to the cell periphery, others contained pyknotic nuclei or none at all. The cytoplasm of the enlarged cells was filled with small, foamy appearing granules (prelipoid) or contained large vacuoles (figs. 2 and 3).

There was complete destruction of the percipient retinal elements. The few remaining nuclei of the outer nuclear layer were almost fused with the inner nuclear layer since nearly the entire outer plexiform layer had disappeared. The inner nuclear layer was the best preserved part of the retina. A number of its nuclei, however, showed an enlarged foamy cytoplasm indicative of lipoid, suggesting scavenger cells, derived from the microglia and possibly the Mueller's cells (figs. 2 and 3).

There was juxtaposition of the external limiting membrane and the pigment epithelium (fig. 3) and gaps in the former permitted entrance of proliferating and migrating pigment epithelial cells into the retina. An increased number of fibers running through the entire thickness of the retina in a direction perpendicular to its expanse, indicated marked gliosis, and Van Gieson stain revealed a thick glial plate at the former site of the first retinal neuron.

The pigment epithelium was present throughout but its cells were irregular in

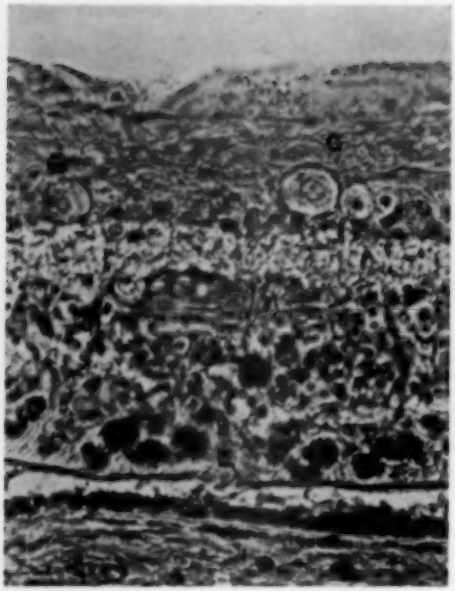

Fig. 3 (Klien). Vacuolated, swollen ganglion cells containing remnants of Nissl granules (G), migration of pigmented cells into retina, holes in external limiting membrane (E). (Toluidin blue.)

size and had uneven pigment content.

The choroid was normal.

The optic nerve was completely atrophic with pronounced neuroglial proliferation. All blood vessels in nerve and retina were apparently obliterated. The sheaths of the optic nerve were normal in appearance.

There was some infiltration with lymphocytes in the axial portion of the nerve along the central vessels. Groups of round cells were encountered also, here and there applied to the inner retinal surface and suspended in the posterior vitreous. This low-grade inflammatory reaction could be considered the counterpart of the leptomeningitis in the central nervous system, which was found in this case and which was thought to be caused by prolonged contact with toxic products of tissue degeneration.

The direct correlation of the clinical and histologic appearance of the fovea centralis, which was possible in this case, is of special interest. The small, punched-out area seen ophthalmoscopically in the center of the macular region was revealed to be caused by the abrupt ending of the gliosed retina at the edge of a slightly pigmented glial plate, apparently derived from the pigment epithelium (fig. 4).

The distinctive clinical appearance of the accompanying optic atrophy indicated by the yellowish gray cast of the optic disc (fig. 1) appears to be the result of the primary degeneration of the third retinal neuron modified by the pronounced gliosis which extended from the retina over the borders of and into the atrophic papilla.

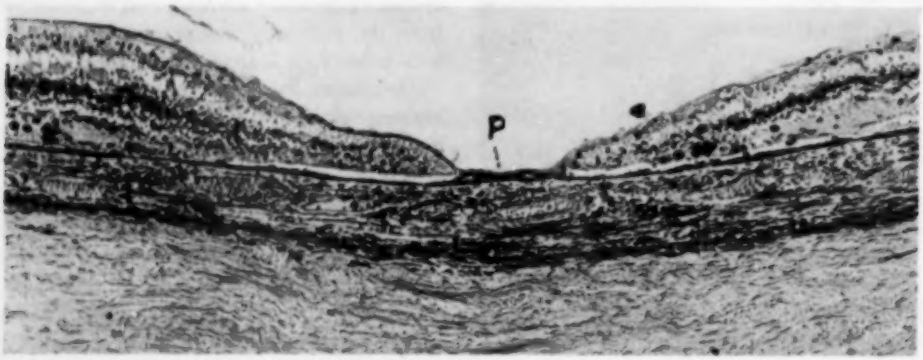

Fig. 4 (Klien). Low-power view of macular area and fovea centralis. Slightly pigmented glial plate (P) occupies fovea centralis, abrupt ending of gliosed retina at its edge caused punched-out appearance clinically.

Comment

In this case the ocular findings constituted the main differential diagnostic problem. The retinal changes indicated a primary lipoid degeneration of the ganglion cells similar to those of the infantile amaurotic idiocy, coexisting with a primary degeneration of the first retinal neuron and secondary gliosis and pigment infiltration of the retina, a combination thought to be characteristic of the juvenile amaurotic idiocy (Sjögren[11]).

The onset of the disease during the second year of life is neither characteristic of the infantile type, which usually begins during the first six months of life, nor of the juvenile type, which has its onset later at the age of five or six years. Since the cerebral lesions were typical of the infantile type this case was classified as one of the less common, delayed infantile amaurotic idiocies.

In view of the associated primary degeneration of the first retinal neuron, and the racial background (non-Jewish), it constitutes a link between the infantile and juvenile types and supports the recent trend of emphasis upon the similarities rather than the dissimilarities between them.

The uncommonly long duration of the disease process in this case accounts for some of its unusual features such as the massive gliosis in brain and retina, the almost complete disappearance of the ganglion cells, the fibrous leptomeningitis, and the chronic inflammatory cells in optic nerve and posterior vitreous, and the complete obliteration of the retinal vessels.

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References

1. Oxford Medicine. New York, Oxford University Press, 1949, ed. 4, v. 2, pp. 214, 408.
2. Rintelen, F.: Histopathologische Veränderungen am Augenhintergrund: die Beziehungen zwischen Tay-Sachs' scher Idiotie und Niemann-Pick's Lipoidosis. Arch. f. Augenh., **109**:322, 1925.
3. Givner, T., and Roizin, L.: Juvenile amaurotic familial idiocy. Arch. Ophth., **32**:39, 1944.
4. Sorsby, A.: Modern Trends in Ophthalmology. New York, Hoeber, 1949.
5. Bielschowsky, M.: Über spätinfantile familiäre amaurotische Idiotie mit Kleinhirn Symptomen. Deutsche Ztschr. f. Nervenh., **50**:7, 1914.
6. Stock, W.: Über eine bis jezt noch nicht beschriebene Form der familiär auftretenden Netzhautdegeneration, etc. Klin. Monatsbl. f. Augenh., **46**:225, 1908.
7. Vogt, H.: Zur Pathologie und pathologischen Anatomie der verschiedenen Idiotie Formen. Monatschr. f. Psychiat. u. Neurol., **24**:106, 1908.
8. Spielmeyer, W.: Ueber familiäre amaur. Idiotien. Arch. f. Psychiat., **40**:1038, 1905.
9. Batten, F. E., and Mayou, M. S.: Family cerebral degeneration with macular changes. Proc. Roy. Soc. Med., **8**:70, 1914-15.
10. Richter, R., and Parmelee, A. H.: Late infantile amaurotic idiocy with marked cerebral atrophy. Am. J. Dis. Child., **50**:111, 1935.
11. Sjögren, T.: Die juvenile amaurotische Idiotie. Lund, Berlingska Boktryckeriet, 1931.

Ophthalmic Miniature

Socrates to Aristodemos:

> Does it not seem to you a work of providence that the creator supplied the visual organ, because it is delicate, with lids as with a door which opens when you need to see, but closes when you sleep; that he fastened lashes to it like a sieve so that the wind may not hurt it; and that, above, like an umbrella, he gave brows so running sweat may never blind you.

Xenophon: Commentaries, 1, 4, 6.

THE PRESENT STATUS OF EPIDEMIC KERATOCONJUNCTIVITIS*

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It has been thought advisable to review critically the present-day knowledge of epidemic keratoconjunctivitis, for, after some years of obscurity, this disease is once more becoming prominent. Epidemic keratoconjunctivitis received a good deal of attention during the early days of World War II when it caused much concern in large industrial plants (table 1) and a virus was isolated from patients in the acute stages of illness.[1] After 1943, however, the disease in its epidemic form seemed to disappear except for two small outbreaks in California in 1947-48,[2] while scientific interest as measured by the number of published articles apparently waned.

The recent recovery of interest is due to a number of factors including the reports of a large epidemic in an industrial plant in Windsor, Canada,[3] and of several small ones associated with certain ophthalmologic clinics in the Midwest,[4] the identification of the reported causal agent as St. Louis encephalitis virus,[5, 6] and the claim of a fresh isolation in Turkey.[7]

The spread of infections seems, at least in some instances, to be associated with examinations in eye clinics, and this has very serious implications for ophthalmologists who might be unwittingly infecting themselves and their patients.

Even more serious is the industrial hygiene aspect. Although the mechanism of spread of shipyard conjunctivitis was never discovered, it is obvious that in large concerns where minor trauma to the eyes is an everyday hazard and the attendances at clinics for treatment runs into the hundreds each day, the possibility of an explosive outbreak due to spread in the first-aid clinic is a real risk. In Chicago on October 15, 1953, a seminar on epidemic keratoconjunctivitis was held by the Eye Bacteriology Group of the American Academy of Ophthalmology. At this seminar much unpublished data were disclosed, some of which are included in this article.

PAST HISTORY

The literature on epidemic keratoconjunctivitis up to 1941 has been fully reviewed by Hogan and Crawford.[8] The disease was first recognized as an entity in Germany toward the end of the last century, and the classical description was given by Fuchs who named it, "keratitis punctata superficialis." Judging by the number of published articles, the disease must have been fairly common at that time; but for many years in the early part of this century little was written about it, giving the impression either that interest in disease had waned or that its incidence was greatly reduced.

There was, however, an epidemic of considerable size in Madras, India, in 1920; and cases were seen every year in that town until another large epidemic was found in the same area in 1930. Reports of new outbreaks in many countries, especially in Asia, then began appearing in the literature. This situation reached a climax in the years 1936 to 1942, after which the occurrences of large-scale epidemics diminished rapidly.

Reported epidemics in the United States, Canada, and Hawaii up to the present are given in Table 1. In addition single cases were described and reported in 1909, 1911, 1922, and a group of cases, which may have included cases of epidemic keratoconjunctivitis, in Michigan, in 1934.

CLINICAL DESCRIPTION

The disease consists of an acute conjunctivitis followed some days later by a keratitis, and the formation of subepithelial corneal opacities.

* From the Communicable Disease Center, Public Health Service, United States Department of Health, Education, and Welfare, Greeley, Colorado.

TABLE 1

PUBLISHED REPORTS OF EPIDEMICS OF EPIDEMIC KERATOCONJUNCTIVITIS IN THE UNITED STATES

Year	Area	Location	No. of Cases	Authority	Journal
1936	San Fernando California	V.A. Hospital (staff patients)	16	Hobson, L. C.	Am. J. Ophth., *21:* 1153–55, 1938
1941	Hawaii	Naval Yards-Pearl Harbor and town	10,000*	Holmes, W. J.	Hawaii M. J., Nov., 1941
1941	San Francisco California	Shipyards and Neighboring town	200	Hogan, M. J. Crawford, J. W.	Am. J. Ophth., *25:* 1059, 1942
1941	Oregon	Shipyard and Neighboring town	600	Rieke, F. E.	J.A.M.A., *119:* 942, 1942
1942	Schenectady	Industrial concern	Approx. 4,000	Perkins, J.	Am. J. Pub. Health, *33:* 1187–98, 1943
	Buffalo	Industrial concern	Some hundreds	Korns, R. F.	
	Rochester	Industrial concern	35	Westphal, R. S.	
1942	Detroit	?	247	Molner, J. C. Cooper, S. L.	Am. J. Pub. Health, *34:* 272–277, 1944
1942	New York	Office infection	80	Sanders, M. Gulliver, F. D. Forcheimer, L. L. Alexander, F. C.	J.A.M.A., *121:* 250–255, 1943
1947–8	California	Office infection I	17	Thygeson, P.	Tr. Am. Ophth. Soc., *46:* 366, 1948
		II	5		
		III	1		
1951	Midwest	Eye clinic I	9	Cockburn, T. A.	Am. J. Ophth., *36:* 1367, 1953
		Eye clinic II	30	Nitowsky, H. Robison, T., Cheever, F. S.	
1953	Windsor Canada	Ford Motor works	At least 600	McGavin, M. F. J. P. Boley	Canadian M. A. J., *69:* 137–140, 1953
	Detroit 1947	Industrial works	Some hundreds	Ormsby, H. L.	

* Authority is Holmes and quoted by Hogan and Crawford.

The conjunctivitis may be mild, moderately severe, or very severe. The inflammation is often follicular in type, and in some epidemics the lower lid is said to have been more affected than the upper. In the more severe cases, there is edema of the conjunctiva which occasionally is very marked. Formation of false membranes is not uncommon. The membrane may be adherent so that attempts at removal may cause slight bleeding. Some of the cases have shown multiple subconjunctival hemorrhages and even petechial hemorrhages in the lids.

The discharge is usually slight and may be yellowish-brown but is never purulent. Tear discharge may be considerable but usually this is associated with the keratitis. Pain and discomfort during the preliminary conjunctivitis is not a presenting symptom. The predominant cells in the eye discharge are monocytes. The preauricular glands on the affected side are usually enlarged as are occasionally the cervical glands.

With the onset of the keratitis the symptoms became more marked, there being usually definite pain, discomfort, and a feeling as though a foreign body was present. Lacrimation may be increased, and photophobia and blurring of the vision are not uncommon. In some cases a fine punctate infiltration is noticed within a few days of onset of the keratitis but normally the first opacities do not appear until one to three weeks after onset.

The cornea may be a little roughened but does not as a rule stain with fluorescein. The opacities are grayish-white infiltrations of cells into the deeper layers of the cornea. They may be only a few in number or there may be hundreds of them. Usually they are described as being nummular or coinlike, 0.5 mm. or less in size, and lying beneath Bowman's membrane; but larger ones discoid in shape and up to four to five mm. have been reported in some epidemics.

The main clinical features of past epidemics in the United States and Hawaii are summarized in Table 2. The severity of the

TABLE 2

CLINICAL DATA

Epidemic	No. of Cases	Severity of Conjunctivitis	% with Bilateral Disease	Degree of Pain	Foreign-Body Sensation	Enlarged Preauricular Glands	Duration of Acute Stage	% with Opacities
San Fernando, Calif. 1936	16	++	13/16	Not severe	?	?	—	Probably 100%
Hawaii 1941	10,000	+++ Chemosis, Conj. hemorrhages	—	Not a prominent feature.	+++	80%	2–3 weeks	Almost 100%
San Francisco 1941	200	+++ Chemosis, hemorrhages	31/125	Discomfort not severe.	+++	75%	Average 13 days.	92/125
Oregon 1941	600	+++ Chemosis,	Usually only one eye	—	+++	—	16–28 days	Majority
Schenectady, N. Y., 1942	4,000	More severe than other epidemics	56.1%	—	++	Almost 100%	—	80%
New York 1942	80	+++ Membrane formation	34/80	+++	++	Most		42/80
Detroit 1942	247	+++ Membranes	—	—	—	—	—	76/219
California 1947–8	90	Membranes 2/21	18/21	+++	—	21/21	—	21/21
Midwest City No. 1, 1951	9	+++ Membrane formation	0/9	+++	++	100%	Average three weeks	8/9
Midwest City No. 2 1951	30	+++ Membranes	About 20%	+++	+++	+	3–5 weeks	6/6

++ Moderately severe.
+++ Very severe.

conjunctival reaction has varied a good deal from epidemic to epidemic. In the United States it has usually been marked, but in some of the epidemics in Germany and the Balkans in 1940-42 it was comparatively mild.

Most commonly the disease is limited to one eye, but in some epidemics bilateral infections have been present in a high percentage of the cases. In Madras in 1928, only 22 cases out of 923 were bilateral. In the San Francisco epidemic of 1941, 31 of 125 cases were bilateral, but in the Schenectady outbreak, 56.1 percent of the cases were bilateral.

Although the majority of patients develop opacities, yet a certain percentage fail to do this. The percentage of patients without opacities varies from 0.0 to 50 percent. Sequelae are mild for, although many opacities persist for one or more years, their presence leads to no marked disability. A diminishing of visual actuity is not uncommon but is rarely of any marked degree. In some of the cases showing adherent membranes symblepharon has resulted.

LABORATORY STUDIES

All investigators are agreed that no organism of a bacterial nature can be shown to be the etiologic agent. Attempts to isolate such an infectious agent by seeding conjunctival scrapings or eye washings to various media have been unproductive. No inclusion bodies in conjunctival scrapings have ever been demonstrated.

Attempts by Wright in 1930 to transmit the infection were partially successful.[9] Using conjunctival scrapings passed through a Kitsato unglazed porcelain filter, he was able to reproduce the disease by inoculation into the blind eyes in five of 13 volunteers suffering from optic atrophy.

Viswalingham[10] was able to transmit infection to four of six similar volunteers, with the inflammation appearing 12 to 36 hours after inoculation. One of the four volunteers developed corneal opacities. On a second

passage to two more volunteers, a mild conjunctivitis was produced in both of the inoculated eyes.

The first isolation of a virus was reported in 1941 by Sanders and Alexander.[1] It had been noted by other workers that injection of apparently sterile conjunctival material into mice produced a transient illness in the animals without killing them.

The technique of growing the suspected virus in tissue culture prior to mouse inoculation was adopted by Sanders and his colleagues in the hope that the preliminary adaptation would cause a more definite effect on the mice. The tissue culture used was a serum ultrafiltrate, a minced embryonic mouse-brain preparation, which was incubated for 48 hours before inoculation to insure sterility.

By such means a virus was obtained from apparently sterile scrapings from a patient with epidemic keratoconjunctivitis, and this virus was maintained for at least 31 generations by transferring the tissue and fluid from infected to fresh cultures every six or seven days. Mouse inoculations were by the intracerebral route and activity titers up to 10^{-6} were recorded.

In the mice, symptoms appeared in two to seven days according to the dilution injected. At first the treated animals were lethargic and had hunched backs and ruffled coats; then often there were tonic and clonic convulsions, or occasionally spastic paralysis; and quite frequently the animals had a peculiar sidling gait suggestive of middle ear infection. Death sometimes followed the onset of lethargy or sometimes was preceded by the whole range of symptoms.

In four rabbits a 1/50 dilution of 22 mouse-passage brain emulsion had no effect for eight days but on the ninth day produced prostration and death of three of them.

In monkeys injected intracerebrally and conjunctivally with the mouse-brain virus, one developed a brain abscess, three showed no effect, and two had vague nervous symptoms before dying in five days.

In guinea pigs and albino rats, no symptoms were produced when these animals were inoculated with the virus by a variety of routes.

The virus was identified by testing for neutralizing antibodies in the sera of convalescent patients, and neutralization up to 10,000 mouse infective doses was obtained, the general range being between 100 and 10,000. Two of the sera were from patients in California, three from a clinic in New York City, and two from an epidemic in New York State. The sera were collected between one and six months after onset of the acute symptoms.

In another series of tests, 15 sera from an outbreak in New York were taken three to six weeks after onset of illness, and all had neutralized 100 to 10,000 mouse doses of virus. In six instances where paired acute-convalescent sera were available, rising titers from zero in the acute specimens to a level of neutralizing 1,000 mouse infective doses in the convalescent specimens taken four weeks after onset were demonstrated.

The epidemic keratoconjunctivitis virus was tested against lymphocytic choriomeningitis virus, Theiler's mouse virus, and herpes virus and no relationship could be demonstrated.

These results would indicate that a virus had been isolated that presumably originated in the eye of an acute case of epidemic keratoconjunctivitis; that a rising titer of antibodies against the virus could be demonstrated in acute cases, and that the disease could be reproduced by inoculation of the virus into the eye of a human volunteer who later developed a rising titer of neutralizing antibodies against the virus. This evidence would support the claim that this epidemic keratoconjunctivitis virus of Sanders is the etiologic agent of the disease.

Conflicting claims have, however, been made with relation to this virus. Sanders and Alexander[1] state that it is not related to herpes simplex virus, while Maumenee and his colleague[11] concluded from their studies

it was practically identical with herpes simplex virus. More recently, Ruchman[5] and Cheever[6] have found that the strain which is now in existence and is labeled epidemic keratoconjunctivitis virus is almost indistinguishable from St. Louis encephalitis virus, while Cockburn, et al.,[4] in two small epidemics in the Midwest, found no neutralizing antibodies against either St. Louis encephalitis or the Sanders epidemic keratoconjunctivitis virus in sera from convalescent patients, and a similar finding was made by Ormsby and Fowle in the Windsor epidemic.[12]

During World War II apparently all strains of epidemic keratoconjunctivitis virus except one were discarded, and all existing strains are derived from this solitary surviving strain, now in the possession of Dr. Braley.[13] Dr. Braley has found that this virus no longer is neutralized by convalescent sera from patients to the extent it had been previously, and suggested that the original virus has been lost or contaminated with St. Louis encephalitis virus.

During the past three years numerous attempts have been made by Ormsby and Fowle[12] to isolate fresh strains by using the Sanders technique, using chiefly material from Toronto and the Windsor epidemic, but all attempts have been unsuccessful. These workers did isolate five strains of virus but these proved to be strains of mouse encephalomyelitis virus closely related to the Theiler TO strain.

Isolation of virus has been reported from Turkey by Sezer.[7] This was accomplished by using an egg technique in which human corneal tissue is implanted on the chorioallantoic membrane and inoculated with the suspected eye material. The virus isolated by this means adapted itself to growth in the egg and produced symptoms in the mouse similar to the Sanders virus. The relationship of this organism to the American disease has not yet been studied.

Another virus was isolated during the war in 1941 by Dresel, et al.[14] in Germany. This virus also produced symptoms in mice similar to those of the Sanders epidemic keratoconjunctivitis virus.

A series of eight isolations have been reported from Japan, according to Braley.[17] Two viruses were recovered from infected eyes and six from patients' sera. The method employed included numerous blind passages in mice, a technique that in this country is regarded as unreliable.

The danger of this technique is illustrated by the experiences of Ormsby and Fowle[12] who, as already described, in their studies using the Sanders technique, in a number of instances recovered viruses which seemed to be latent mouse viruses that were reactivated by the manipulations. Additional evidence as to the nature of the organism recovered in Japan is needed before comment can be made.

To sum up the situation in this country at the present there is no virus available that can be regarded with confidence as the etiologic agent of epidemic keratoconjunctivitis. The relationships of those viruses reported abroad to epidemic keratoconjunctivitis must be considered doubtful until further more conclusive evidence is provided on their natures.

Epidemiology of epidemic keratoconjunctivitis

As a worldwide disease

Epidemic keratoconjunctivitis has been reported from Europe, North America, and Tasmania, and in many countries of Asia, including India, China, Japan, and Malaya, Hawaii, the United States, and Canada. It is a temptation to speculate that the infection spread eastward from an Asiatic focus to the Naval dockyards at Hawaii, then to California, and finally to New York, but this idea has nothing to support it beyond the sequence of events.

The method of spread in parts of the world other than the United States is obscure. In Madras where 20,000 patients are treated as out-patients at the eye hospital,

TABLE 3

PATIENTS WITH EPIDEMIC KERATOCONJUNCTIVITIS ATTENDING THE EYE HOSPITAL IN MADRAS, 1930 (WRIGHT)

Age Group	Male	Female	Total	% of Total
1– 9	3	4	7	0.8
10–19	113	43	156	16.9
20–29	413	55	468	50.7
30–39	167	27	198	21.0
40–49	55	9	64	6.9
50–59	16	11	27	2.9
60–69	5	2	7	0.8
	772	151	923	

small numbers of epidemic keratoconjunctivitis cases are seen every year; but in 1920 and 1928 there were substantial epidemics involving thousands of people, and Wright[9] expresses the opinion that the disease was common at this time in other parts of India, also. Apparently it affected all age groups, as indicated in Table 3. The differences between the number of cases in the age groups are not significant in that the majority of patients attending the eye hospital were adult males. All races were involved in the epidemic.

In the Calcutta epidemic in 1933, Sanyal[18] reported on 175 cases in persons living in close contact in families, and in hostels among students sharing the same rooms. There appeared to be no racial factor involved, and all ages, and people of differing social standings were affected.

In Malaya in epidemics in 1935-38 it was a similar story of all ages, races, social standings, and both sexes being involved. The age distribution of cases was similar to that in Madras (table 4).

To sum up, the evidence from abroad is too scanty to permit conclusions although it is fairly evident that transmission inside a family group or among people sharing the same room would be well established. The observation of Viswalingham[10] that susceptibility to infection was "great in eyes that were convalescing after trauma, either surgical or otherwise," seems significant in view of American experiences.

AS AN INFECTION IN INDUSTRIAL PLANTS

The reported accounts of epidemics in industrial plants show two things quite clearly: (1) In the area the earliest cases were in men working in parts of the plants where minor eye injuries are common, and (2) that later cases occurred among the families, contacts, medical attendants, and people in the town surrounding the industrial plants. This sequence was seen in the epidemics in Hawaii, Oregon, San Francisco, and Schenectady.

There can be little doubt that transmission among people living together does occur quite frequently, just as happened in the outbreaks in India. However, the infectiousness of the organism does not appear to be high, and in the Schenectady epidemic it was estimated that among the families with an infected adult, only four percent of the exposed people became ill. This figure was approximately the same as for the total group of factory workers since only small sections of the factory population developed symptoms, but the incidence rates in the affected parts of the factory must have been much higher. Obviously, with an organism of so low an infectivity, the method of transfer from one person to another must be fairly direct.

The method of transmission inside the factory is not known. It has been remarked repeatedly that the workers who are most liable to develop the disease are those ex-

TABLE 4

AGE DISTRIBUTION OF CASES OF EPIDEMIC KERATOCONJUNCTIVITIS IN MALAYA 1935–38 (VISWALINGHAM)

Age Group	No. of Cases	% of Total
0– 1	76	2.1
1– 9	824	22.8
10–19	890	24.6
20–29	872	23.8
30–39	518	14.4
40–49	218	6.0
50–59	92	2.6
60–69	26	0.7
70–79	5	0.1
	3521	

posed by the nature of their work to injury by foreign bodies, flash burns, or bright lights, and the question often has been raised as to whether the injury could be responsible for initiating the infection. The problem is complicated in that a prominent symptom of the disease is a feeling that a foreign body is present in the eye.

The only actual data available are for the Schenectady outbreak when 47 percent of the persons affected in the factory gave a history of having attended the factory clinic immediately prior to the onset of the illness for some minor eye ailment. No accurate data are available but it is understood that in the first San Francisco epidemic, many of the welders who developed the disease had not attended the clinic prior to the onset of illness.

Injury to the eye could initiate the disease in three ways:

1. A latent infection could be activated in the same manner as a circumoral herpes infection is stimulated by exposure to ultraviolet light.

2. The trauma could make the eye particularly liable to damage to a transient infection that otherwise would cause no pathology.

3. The clinic may be the focus of infection; and the trauma of the eye merely takes the person there for examination where he becomes infected.

Sufficient data to allow for further comments on these three points have not been published but it is hoped that attention will be paid during future epidemics to the possibility that the primary source of infection is in the factory clinic itself. Other possible means of spread are by the use of common towels, although there is no evidence to support this. Medical attendants and physicians are particularly liable to become affected, presumably from treating some case and transmitting the infection to their own eyes by means of their own contaminated fingers.

As an infection of eye clinics

It has been known for some years that physicians themselves may transmit the infection from patient to patient in the course of eye examinations, outbreaks having been described from New York[15] and California.[2] At the recent Eye Bacteriology Group meeting at Chicago, this matter was discussed at length, and it was found that practically all the members present were aware of unreported outbreaks of this nature during the past 10 years.

Those mentioned at this meeting and those reported to me personally at different times cover the whole country including Kansas City, Iowa, Cincinnati, Montana, San Francisco, Los Angeles, San Pedro, New York, Denver, Chicago, and Philadelphia. In many instances the physician himself became infected. Beyond question, many more outbreaks have occurred than published accounts would indicate.

It was the opinion of the group members that the means of transfer of the virus could be the fingers of the attendant, infection of the various eye solutions, or instruments such as the tonometers used for measuring eye tensions. Ophthalmic solutions and dropping bottles have been incriminated several times in the past, with suggestions that the infective agent can survive for long periods in these fluids. In the midwest city Epidemic I (see tables 1 and 2), it was shown how a small unsuspected infection could be maintained over a period of months in a glaucoma clinic, the number of patients being small but infection being passed from one to the other by a tonometer.

The danger is particularly acute when patients travel from place to place. The greatest danger comes from eye clinics attached to teaching hospitals, for to them patients are continually being sent by physicians in small towns, patients who may be in an infective state. Should an epidemic occur in the teaching hospital, then other patients may set off a number of smaller epidemics upon their return to the towns in which they live.

By this means the infection could be spread over the whole country from the one focus.

Interepidemic maintenance of infection

The reservoir of the virus between epidemics is not known but there are at least five possibilities:

1. Serial passage from patient to patient, as that described in a glaucoma clinic in a midwestern town, the cases being so few as to go unrecognized as epidemic keratoconjunctivitis in epidemic form.
2. Cases of infection existing without keratitis and which may cause only a mild conjunctivitis. Such cases are not diagnosed as epidemic keratoconjunctivitis, but are capable of causing the classical epidemic disease under the proper circumstances.
3. The agent normally present in an inapparent form in the population and becoming activated by certain stimuli in the same way as herpes simplex.
4. Symptomless carriers.
5. An animal reservoir.

For the present the last two possibilities can be dismissed with the remark that, until some way of isolating and identifying the causal organism has been devised, there is no way of testing them.

The first possibility is a very likely one, the chronic nature of the infection permitting a continual sequence of cases to exist. However, it has been my experience in a small number of outbreaks that the initial case that triggers the outbreak appears to have had no connection with any other known case. Also, ophthalmologists often see isolated cases with conjunctivitis and corneal lesions that appear to have no relationship to any other such illness either within the family or at work. It is difficult to account for these initial or sporadic cases.

The second possibility is also very likely as suggested by Sanders and Alexander[19] in connection with the New York outbreak. It is known that such atypical cases occurring sporadically would not be diagnosed as epidemic keratoconjunctivitis and until the causal agent has been identified and a simple test made available, the accurate labeling of such sporadic affections will remain almost impossible.

The suggestion that such cases of epidemic keratoconjunctivitis without the keratitis do exist and can initiate classical epidemics made first by Sanders, et al.,[19] has been confirmed by the findings in the midwest epidemic[16] in which the initial case was atypical and only diagnosed in retrospect, as well as in a later instance reported by Cockburn and Danielson.[15] In this later outbreak, the initial patient had a mild chronic conjunctivitis for five weeks, but developed classical epidemic keratoconjunctivitis seven days after treatment with cortisone.

In the Windsor epidemic[3] it was specifically stated that the cases at the beginning and end of the epidemic were mild and atypical, and Ormsby and Fowle[12] further report that such cases continue to be seen in the Toronto region but having no obvious connection with classical cases of epidemic keratoconjunctivitis it is not possible as yet to lable them as epidemic keratoconjunctivitis. In view of this evidence it would seem quite probable that the reservoir of causal agent should be in such mild chronic atypical cases.

It has been suggested[15] that cortisone might activate such an atypical infection and convert it into the more severe classical type. Up to the moment the evidence for this is based on one case and nothing more can be added until further data becomes available.

Other forms of keratoconjunctivitis

1. Instances of conjunctivitis without keratitis that appear to be exactly the same as those described above are quite common. In Kansas City in one month in 1952, my colleague, Dr. H. Nitowsky, and I with the co-operation of the ophthalmologists and other physicians of the town, saw nearly 300 cases that, if seen only in the initial phases of the illness during an epidemic, would have been diagnosed as epidemic keratoconjunctivitis.

It was also obvious at that time that many hundreds of cases of a mild nonpurulent

conjunctivitis were present, particularly in school children. The majority of these were never seen by physicians, and those who did seek medical advice consulted general practitioners or pediatricians, not ophthalmologists.

Undoubtedly infectious conjunctivitis is much more common than is generally supposed. There is, of course, a well-recognized entity, Beals' conjunctivitis, which is not followed by corneal opacities, as well as conjunctivitis due to Newcastle disease virus which is primarily an infection of poultry but which can cause an acute human conjunctivitis. Both of these have superficial resemblances to the conjunctival stage of epidemic keratoconjunctivitis but produce no opacities.

2. Herpes simplex is said to produce a picture almost indistinguishable from epidemic keratoconjunctivitis in the conjunctivitis stage, although the corneal lesions are dissimilar, and there have been several isolations of the herpes-simplex virus from such cases.

3. Greeley disease is a recently described entity in which there is moderately severe conjunctivitis followed in some instances by small corneal opacities and associated with systemic symptoms, muscle pains, and vesicular pharyngitis. No specific organism has yet been associated with this disease.[20]

It is well known that leptospiral infections of the eye occur, producing various pathologies including conjunctivitis. The diagnosis of Leptospira canicola infection has been made retrospectively in an epidemic closely resembling those of "Greeley disease."[16]

Discussion

Epidemic keratoconjunctivitis is a reportable disease in 22 states but the numbers of cases are extremely meager. It is obvious that this is due to lack of reporting and not to the absence of the disease, for practically every ophthalmologist with whom the matter has been discussed has known of an epidemic in his area. If it is accepted that many of the cases without keratitis occur without being diagnosed, then the total yearly occurrence of the disease must run into substantial figures.

The following points would appear to be quite clear:

1. Epidemic keratoconjunctivitis is a worldwide disease of considerable potentialities.

2. In the United States it seems to be epidemic, being spread either in association with eye clinics or between people living in intimate contact with one another. At times it becomes epidemic in industrial plants, where a large proportion of the cases occur in workers exposed to minor eye injuries.

3. A satisfactory strain of the causal agent is not available in this country and little fundamental work on the epidemiology of the disease can be done until one is produced.

4. Prevention of transmission of infection in eye clinics is a responsibility of both ophthalmologists in practice and persons responsible for the maintenance of first-aid stations in industrial plants. A special responsibility falls on the larger eye clinic associated with medical schools, for an epidemic in such a place might spread infection over the whole country.

5. The large epidemics radiating from industrial plants could be explained by available data. The initial infection could be introduced to the factory's first-aid clinic and the infection then transmitted to patients attending for minor eye ailments; the patients would carry the infection to their homes, and their families would in turn develop the disease.

Secondary outbreaks would be set up both in the town, when affected members of the families consulted ophthalmologists, and in the industrial plant where transmission would occur in suitable places as, for example, among the night watchmen of the Schenectady epidemic who lived closely together while on duty. Eventually, as the number of cases accumulated outside of the plant, some employees would be infected there and carry the disease back with them to their work places.

Urgently required is a study aimed at clarifying the whole problem on a national basis. Now that doubt has been cast on existing strains of the epidemic keratoconjunctivitis virus, the present position is not much in advance of that of a decade ago when the disease first became a problem in the United States. The only real progress made is in the recognition of the spread by ophthalmologists themselves.

The fundamental points to be settled are:

1. The nature of the etiologic agent.
2. Production of a simple serologic test to be used for diagnostic purposes.
3. The clarification of the etiologies of the large group of conjunctivitis infections without keratitis.
4. Methods of transmission in the industrial plant, the eye clinic, and the home.
5. The interepidemic reservoir of the infection.
6. Re-evaluation of techniques used by ophthalmologists that might transmit infections.
7. Treatment of cases.

A program of study to deal with these points would require the collaboration of large numbers of practicing ophthalmologists, laboratory workers, epidemiologists, and industrial medical officers; until this is undertaken, the problem will continue to exist and the disease will be a threat to the public in general and especially to all those involved in work with eyes.

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References

1. Sanders, M., and Alexander, R. C.: Epidemic keratoconjunctivitis: I. Isolation and identification of a filterable virus. J. Exper. Med., **77**:71 (Jan.) 1943.
2. Thygeson, P.: The epidemiology of epidemic keratoconjunctivitis. Tr. Am. Ophth. Soc., **46**:366-385, 1948.
3. McGavin, M. F., Boley, J. P., and Ormsby, H.: Epidemic keratoconjunctivitis. Canadian M. A. J., **69**:137-140, 1953.
4. Cockburn, T. A., Robison, T., Nitowsky, H., and Cheever, F. S.: Epidemic keratoconjunctivitis: A study of a small epidemic. Am. J. Ophth., **36**:1367-1372, 1953.
5. Ruchman, I.: Relationship between epidemic keratoconjunctivitis and St. Louis encephalitis viruses. Proc. Soc. Exper. Biol. & Med., **77**:120-125, 1951.
6. Cheever, F. S.: A possible relationship between viruses of St. Louis encephalitis and epidemic keratoconjunctivitis. Proc. Soc. Exper. Biol. & Med., **77**:125-129, 1951.
7. Sezer, F. N.: Cultivation of virus of epidemic keratoconjunctivitis on chorioallantoic membrane of fertile eggs. Arch. Ophth., **49**:293-302, 1953.
8. Hogan, M. J., and Crawford, J. W.: Epidemic keratoconjunctivitis (superficial punctate keratitis, keratitis subepithelialis, keratitis maculosa, keratitis nummularis): With a review of the literature and a report of 125 cases. Am. J. Ophth., **25**:1059 (Sept.) 1942.
9. Wright, R. E.: Superficial punctate keratitis. Brit. J. Ophth., **14**:257-291; 595-601, 1930.
10. Viswalingham, A.: Epidemic superficial keratitis in Malaya. **25**:313-324, 1939.
11. Maumenee, A. E., Hayes, G. S., and Hartman, T. L.: Isolation and identification of the causative agent in epidemic keratoconjunctivitis (superficial punctate keratitis) and herpetic keratoconjunctivitis. Am. J. Ophth., **28**:823-839, 1945.
12. Ormsby, H. L., and Fowle, A. M. C.: Epidemic keratoconjunctivitis. Am. J. Ophth., **38**:490-495, 1954.
13. Braley, A. E.: Presentation, Eye Bacteriology Club, Chicago, 1953.
14. Dresel, E. G., Weineck, E., and Meding, B.: Ueber die Virusnatur der epidemischen Keratoconjunctivitis. Klin. Wchnschr., **20**:999, 1941.
15. Cockburn, T. A., and Danielson, R. W.: Am. J. Ophth., **38**: 1954.
16. Cockburn, T. A., Vavra, J. D., Dann, R., Peterson, L. J., Spencer, S. S., and Reinhard, K. R.: Human leptospirosis associated with a swimming pool diagnosed after 11 years. Am. J. Hyg., In press.
17. Braley, A. E.: Annual reviews: The lids, lacrimal apparatus, and conjunctiva. Arch. Ophth., **51**: 91-137, 1953.
18. Sanyal, S.: Epidemic superficial keratitis, Am. J. Ophth., **16**:390-393, 1933.
19. Sanders, M., Gulliver, F. D., Forcheimer, L. I., and Alexander, R. C.: Epidemic keratoconjunctivitis: Clinical and experimental study of an outbreak in New York City. J. A. M. A., **121**:250-255, 1943.
20. Cockburn, T. A.: An epidemic of conjunctivitis in Colorado associated with pharyngitis, muscle pain, and pyrexia. Am. J. Ophth., **36**:1534-1539, 1953.

EPIDEMIC KERATOCONJUNCTIVITIS*

Observations on four cases

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In September and October, 1953, there was a small outbreak of four cases of epidemic keratoconjunctivitis in Denver, the first patient being a salesman and the other three consisting of his wife, his eye physician, and the physician's wife. There is a clear pattern of spread from the initial case to the other three and in this paper the question of interest lies in the origin of the primary infection and the method of treatment of the secondary cases.

The epidemiology of epidemic keratoconjunctivitis is obscure and the location of the infectious agent between epidemics is unknown. In this instance, the initial patient had a mild chronic conjunctivitis without keratitis for about five weeks before the onset of classical epidemic keratoconjunctivitis, and this apparently was related to the topical application of cortisone. If the observations in this one case were confirmed, a possible mechanism for the interepidemic maintenance of the agent could be postulated. There is no specific treatment for epidemic keratoconjunctivitis, but in this instance the secondary cases were treated with serum from the first patient.

Chronology

This initial patient, a salesman, aged 58 years, travels almost continually, and in August, 1953, was moving almost daily from town to town. On August 6th, three days out of Denver, he developed an acute conjunctivitis in his right eye, which was troublesome enough to make him consult an ophthalmologist in Louisville, Kentucky. The eye was deeply red, with moderate epiphora but apparently there was no purulent discharge. No foreign body was found, and, as the condition was not particularly painful, the patient continued his traveling and business.

After about a week, the redness subsided to a large degree, but the watering continued to be annoying, so that on his return home, the patient visited an ophthalmologist on September 9th. His eye condition was diagnosed as being an allergy and was treated with cortisone topically. On September 17th he developed classical epidemic keratoconjunctivitis. The patient's wife and the physician became ill with similar symptoms on September 27th—10 days after the initial patient and the physician's wife became ill on October 13th.

Clinical description of cases

Case 1

Patient D. H., the initial patient, on September 9th was found to have a mild conjunctival injection of the affected eye and moderate epiphora, although the tear duct was patent and fluid was syringed freely into the nose. Since he had had a history of recurrent corneal erosion in the period of January to June, 1949, his cornea was carefully examined on this occasion with the slit-lamp by three ophthalmologists, but no pathologic condition of the cornea was discovered. As treatment, 0.5-percent cortisone-acetate emulsion was prescribed, on the basis that he might have an allergy to some drug used during the previous month.

On September 17th, the symptomatology

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was greatly increased, with much pain and discomfort, and there was greater redness of the conjunctiva, marked capillary dilatation of the upper tarsus, and marked conjunctival edema. There was no purulent discharge, no opacity in the cornea, and no staining with fluorescein. Treatment was changed to gantrisin solution topically and penicillin and streptomycin intramuscularly.

On September 24th, a small corneal epithelial erosion that stained with fluorescein was discovered, and the preauricular glands were enlarged and tender. Two days later numerous corneal erosions and subepithelial infiltrates were present.

A direct smear was taken, and a general pathology laboratory reported the presence of a few pus cells, many epithelial cells, and rare possible inclusion bodies of initial and elementary type. A culture grew hemolytic and nonhemolytic Staphylococcus aureus.

The duration of the acute illness was about four weeks, the eye still being injected on October 9th, and opacities were still present in the cornea in November.

Case 2

R. D., ophthalmologist, on September 27th noted a slight conjunctival redness of his left eye, and by the next day this had increased to a marked conjunctivitis with much discomfort. The condition rapidly became worse, with much watering of the eye, marked edema of the conjunctiva, and a feeling as of cockleburs being present under the lids; the symptoms lasted for 12 days. Numerous corneal opacities were first noted on October 7th—10 days after onset. A mild inflammation of the right eye commenced on October 7th but this was transient and no opacities appeared in the cornea. Swelling and tenderness of the corresponding preauricular gland were present. Mild generalized aching was noted.

By October 14th, the corrected vision had dropped to 20/200 but had recovered to the normal of 20/15 by November 10th. In late November and early December, the patient noted rings around lights. Three months after onset, 12 opacities still remained in the cornea, two of them being on the pupillary border.

Case 3

C. H., wife of the initial patient, became ill on September 27th with an acute conjunctivitis in the left eye accompanied by watering and a feeling as of a foreign body being present in the eye. The illness was brief, and, by October 20th, an eye examination revealed neither conjunctivitis nor opacities.

Case 4

L. D., the ophthalmologist's wife, first developed symptoms on October 13th, 16 days after her husband. There was stiffness of the lids, redness, epiphora, and acute discomfort. But the duration of the illness was short and the eye was clear by October 23rd. The cornea was not observed until the middle of November when two small opacities were seen with the slitlamp.

These cases were treated by Dr. J. C. Long and Dr. G. S. Tyner, and seen in consultation by Dr. George Filmer of Denver and Dr. A. E. Braley of Iowa City, Iowa.

Treatment

There is no specific treatment of epidemic keratoconjunctivitis but, at the suggestion of Dr. Braley, it was decided to use serum from the first patient as eyedrops to treat the others. It is not known how quickly antibodies against the causal agent appear after the onset of illness, but rising titers against the Sanders epidemic keratoconjunctivitis virus have been demonstrated in 17 days.

Serum was obtained from the first patient on September 28th—11 days after the onset of symptoms, and again about five days later, and drops of this serum were applied topically to the affected eyes of all secondary cases.

Patients R. D. and C. H. received the 11-day and 16-day serum; Patient L. D. was

treated only with the 16-day serum. The results of this treatment were equivocal, for while C. H. and L. D. had only mild illnesses that soon cleared up, R. D. had a particularly severe attack that did not seem affected by the serum. However, his acute phase lasted only two weeks; whereas, a case of this severe type usually continues for much longer than that, so it is possible that the serum had some effect.

Discussion

A point of interest in this small outbreak is the origin of the infection in the first case. As in all other outbreaks reported in the literature, there appears to be no connection between the initial case and any prior case, for no such illness had been seen in the ophthalmologist's office for many years, and the patient stated that none of his friends or business colleagues had had an inflamed eye.

The location of the causal agent of epidemic keratoconjunctivitis between epidemics is unknown but the likelihood of the existence of mild atypical cases must be seriously considered. This idea was originally suggested by Sanders, et al.,[3] in connection with an outbreak in an ophthalmologist's office in New York in 1942, in which the first three cases were spread over a period of many weeks and all were mild and atypical. Similarly, the first case of the Midwest outbreak in a glaucoma clinic[5] had no opacities and was diagnosed only in retrospect, while in the large epidemic in Windsor, Canada,[8] it was specifically mentioned that at the beginning and end of the series of cases, the illnesses were mainly benign and atypical. Such cases continue to appear in the Toronto area, but being sporadic ones and having no apparent connection with any of the classical types, the exact diagnosis of these remains in doubt.[4]

It is with this background that the relatively mild preliminary conjunctivitis of the initial patient becomes of importance, for it is possible that this represented a chronic, relatively quiescent form of epidemic keratoconjunctivitis infection existing for many weeks until flared up by the application of cortisone.

The patient had a moderatively severe conjunctivitis early in August, the acute stage lasting a week and being replaced by a mild but annoying chronic condition. Seven days after the commencement of topical cortisone treatment, the typical signs and symptoms of epidemic keratoconjunctivitis developed and the diagnosis was confirmed by the appearance later of three secondary cases.

Of course, the transmission of infection to this patient could have occurred in the office during the examination in September, and the seven-day delay in appearance of symptoms could correspond to the incubation period of the illness. However, the absence of other cases in the office at the time is against this explanation, for it would be necessary to postulate the presence of a symptomless carrier as the source of infection.

No tonometer was used in any of the four patients. In the absence of any other demonstrable source, the most likely possibility is that the mild chronic conjunctivitis was, in fact, due to an infection with the causal agent of epidemic keratoconjunctivitis. Confirmation of such an observation would lead to serious consideration of the theory that the interepidemic reservoir of epidemic keratoconjunctivitis is in such mild, chronic atypical cases.

The suggestion that this condition was activated by cortisone is in line with recent animal experiments. It has been found that the tissue reactions to organisms are so modified by cortisone as to allow normally harmless saprophytes to invade them with impunity, although at first the inflammatory processes may be mild or absent.[5] Although there may be ameliorations of symptoms, bacteriologic studies indicate no apparent improvement or even definite impairment in the capacity of the animal to dispose of the offending agent. Use of cortisone in rabbits

inoculated with herpes simplex virus into the cornea have given doubtful results[6-8] with the general impression that if the course of the infection was influenced, it was in the direction of increased severity of the pathologies.

There is, therefore, a definite possibility that local applications of cortisone to the eye might actuate latent organisms or cause an exacerbation of an already existing chronic infection, although at first its influence might be to modify or suppress any inflammatory processes. It is suggested that the present case represents such an instance, and it is hoped that if others like it do occur that they will be reported. Confirmation of such a finding would lead to important deductions in the epidemiology of epidemic keratoconjunctivitis and in the treatment with cortisone of eye infections in general.

No conclusions can be drawn from the treatment of the secondary cases with serum from the initial case, and the method is described solely in the hope of encouraging further work along these lines. The lack of response of this disease to therapeutic agents is well recognized but the use of immune serum had not been properly explored up to the present. In this instance it is unlikely that high titer antibodies had developed in the initial patient at the time the blood was drawn, but no other source was available.

It should be possible to prepare in advance for further cases by collecting immune sera from convalescent patients, of whom there are substantial numbers throughout the country, as, for example, those hundreds in the Windsor, Ontario, epidemic, 1952-1953.

This serum could be used either topically or intramuscularly, and supplies could be stored at strategic places and made available to physicians as required. However, there is the well-known and definite danger of the recipients of the convalescent sera being infected with serum hepatitis virus and other agents, so that it would be safer to process the sera to prepare the gamma-globulin fraction. Unfortunately this is a lengthy and expensive process that would be justified only if the demand was great enough to cover the cost, and is beyond the capacities of most independent investigators. It is urged that the matter be studied further through the collaboration of a number of groups.

Summary

1. A case of epidemic keratoconjunctivitis followed by three secondary cases is reported.

2. The onset of acute symptoms in the initial case was preceded by a chronic conjunctivitis that may have represented an atypical form of the disease. It is suggested that the interepidemic reservoir of the causal agent may be found in such cases.

3. The topical application of cortisone may have been related to the onset of the acute symptoms.

4. Serum from the initial patient was used to treat the three secondary cases.

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References

1. Sanders, M., Gulliver, F. D., Forchheimer, L. L., and Alexander, R. C.: Epidemic keratoconjunctivitis. J.A.M.A., **121**:250-255, 1943.

2. Cockburn, T. A., Robison, T., Nitowsky, H., and Cheever, F. S.: Epidemic keratoconjunctivitis: A study of a small outbreak. Am. J. Ophth., **36**:1367-1372, 1953.

3. McGavin, M. F., Boley, J. P., and Ormsby, H.: Epidemic keratoconjunctivitis. Can. M. A. J., **69**: 137-140, 1953.

4. Ormsby, H., and Fowle, A. M. C.: Epidemic keratoconjunctivitis. Am. J. Ophth., **38**:490-495, 1954.

5. Kass, H., and Finland, M.: Adrenocortical hormones in infection and immunity. Ann. Rev. Microbiol., **7**:361-388, 1953.

6. Hallet, J. W., Leopold, I. H., and Steinwetz, C. G.: The effect of systematic cortisone and ACTH in experimental herpes simplex keratitis. Arch. Ophth., **46**:268-270, 1951.

7. Ormsby, H., Dempster, G., and van Rooyen, C. E.: The effect of cortisone in experimentally induced herpetic keratitis of the rabbit. Am. J. Ophth., **34**:1687-92, 1951.
8. Thygeson, P., Geller, H. O., and Schwartz, A.: The effect of cortisone on experimental herpes simplex keratitis of the rabbit. Am. J. Ophth., **34**:885-888, 1951.

EPIDEMIC KERATOCONJUNCTIVITIS*

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In the spring of 1951 an outbreak of epidemic keratoconjunctivitis occurred at the Ford Motor Plant in Windsor, Ontario, as reported by McGavin, et al.[1] During the first four weeks of the epidemic the clinical manifestations of the disease were mild and consisted of marked tearing in the presence of a follicular conjunctivitis and slight enlargement of the preauricular nodes. None of these early cases were severe, and no corneal opacities developed.

By the third week of the epidemic, new cases developed a more acute conjunctivitis, often associated with pseudomembranes. After the fourth week many developed typical subepithelial opacities. Five months later, as the epidemic waned, nearly all cases were mild, and very few opacities developed. Of a total of 549 patients who reported for treatment at the plant hospital, only 89 developed corneal opacities which could be seen by focal illumination with the aid of a binocular loupe.

During the winter of 1951-52 a number of patients with follicular conjunctivitis, tearing, and preauricular adenopathy were studied in the eye bacteriology laboratory at the University of Toronto. These patients had a lymphocytic exudate in conjunctival scrapings, marked lacrimation, and preauricular adenopathy. They resembled the patients seen in the Windsor epidemic. Only one of these Toronto cases developed typical corneal subepithelial opacities, another had pseudomembranes, and one other had subconjunctival hemorrhage.

These observations are in keeping with those of Hogan and Crawford,[2] who first named the disease epidemic keratoconjunctivitis in 1942, and described 125 cases, 17 of which developed macular lesions in the substantia propria (corneal opacities) and 17, pseudomembranes. However, since all are not agreed that during epidemics of epidemic keratoconjunctivitis, mild atypical cases are seen which are less acute, fail to develop pseudomembranes and show no evidence of keratitis, the possibility that some other disease entity may exist, should be kept in mind.

Two separate studies were made of these Windsor and Toronto cases of epidemic keratoconjunctivitis. In one, convalescent sera from a large number of the patients were tested for the presence of antibodies against the Sanders-Braley strain of epidemic keratoconjunctivitis virus,† to determine whether there was any relationship between the virus isolated by Sanders and Alexander[3] in New York, and the virus causing epidemic keratoconjunctivitis in Ontario. In the other, eye washings and conjunctival scrapings from a few of the patients were taken, and an attempt was made to isolate the epidemic keratoconjunctivitis virus, using essentially the tissue culture technique described by Sanders and Alexander.[3]

1. Relationship of the Sanders-Braley virus to the Ontario epidemic

Introduction

During the epidemic at the Ford Motor Plant in Windsor, Ontario, samples of blood

* Provided by Dr. Alson E. Braley, University of Iowa Medical School.

† From the Department of Ophthalmology and the Connaught Medical Research Laboratories (Virus Division), University of Toronto.

were taken from 300 individuals who had recovered from epidemic keratoconjunctivitis, 53 of these being opacity cases. The blood was taken within the first six months after the onset of the epidemic. Seventeen months later, a second sample of blood was obtained from 36 of these same opacity cases and 48 nonopacity cases. The serum was extracted from the blood and stored at −60°C.

At the time of writing, serologic studies have only been done on serum from the opacity cases. In all, 61 sera were used. Individual neutralization tests were done on two-phase sera from 23 cases, first-phase serum from one case, and second-phase serum from four cases. In addition, first-phase sera from 10 cases were pooled and used in a single test. Altogether serum from 38 cases showing corneal opacities was tested for neutralizing antibodies to the Sanders-Braley virus.

Blood was also obtained from each of the five Toronto patients with epidemic keratoconjunctivitis, both during the disease and at intervals up to one year later. Serologic tests were carried out with two-phase sera from three of the Toronto patients, and one-phase serum from the other two patients, giving a total of eight sera tested. These sera selected for testing were taken eight to 12 months after the onset of the disease.

Three sera known to neutralize the Sanders-Braley epidemic keratoconjunctivitis virus were used as controls in testing the above sera from the Windsor and Toronto patients for the presence of antibodies against this strain of virus: (1) Human serum known to have a log neutralization index of 2.9 to the Sanders-Braley epidemic keratoconjunctivitis virus provided through the kindness of Dr. T. E. Sanders (personal communication); (2) hyperimmune rabbit-serum prepared in our laboratory against the Sanders-Braley epidemic keratoconjunctivitis virus; (3) hyperimmune rabbit-serum to the St. Louis encephalitis virus (Webster strain), provided by Dr. F. S. Cheever, having approximately a log neutralization index of 3.0.[4]

A negative control of inactivated (56°C. for 30 min.) normal rabbit-serum or human baby-serum was carried out with each experiment.

Procedure

Ten-fold dilutions of a 10-percent suspension of infected mouse-brain were made in gelatin-buffered saline. To each of these dilutions of the virus was added an equal amount of undiluted serum and the virus-serum mixtures were incubated in a 37°C. water-bath for two hours. Then 0.03 ml. of each dilution was inoculated intracerebrally into five male albino mice weighing 14 to 16 gm. The LD_{50} was calculated.*

Results

No neutralization of the Sanders-Braley epidemic keratoconjunctivitis virus was obtained with any of the eight sera from the Toronto cases, or with 60 of the 61 sera tested from the Windsor epidemic. One first-phase Windsor serum gave a doubtful positive reaction, but unfortunately there was not enough serum to repeat the test. The second-phase serum from the same patient was definitely negative. It may be concluded, therefore, that all sera tested were negative.

The results of neutralization tests with three of the Toronto sera, four Windsor sera, and the three positive control sera are presented in Table 1.

2. Attempts to isolate virus of epidemic keratoconjunctivitis

Procedure

Fluid tissue cultures in flasks containing chopped embryonic-mouse-brain as tissue were used. Either Morgan and Parker's synthetic medium No. 199[5], or Hank's salt solution containing Simms' ox-serum ultrafiltrate served as medium. In most instances the cultures were inoculated with 0.1 ml. of

* Method of Karber.

TABLE 1

NEUTRALIZATION TESTS WITH THE SANDERS-BRALEY EPIDEMIC KERATOCONJUNCTIVITIS VIRUS AND CONVALESCENT SERA FROM EPIDEMIC KERATOCONJUNCTIVITIS CASES IN ONTARIO

Patient	Months after Illness Blood Taken	Test Serum LD_{50}	Normal Rabbit-Serum LD_{50}	Log Neutralization Index
Toronto Cases				
L.	12	$10^{-6.5}$	$10^{-6.4}$	—
M.	9	$10^{-6.5}$	$10^{-6.7}$	—
R.G.	8	$10^{-6.7}$	$10^{-6.4}$	—
Windsor Cases				
No. 232	3	$10^{-6.3}$	$10^{-6.3}$	—
No. 232	20	$10^{-6.9}$	$10^{-6.3}$	—
No. 233	3	$10^{-6.1}$	$10^{-6.3}$	—
No. 233	20	$10^{-6.3}$	$10^{-6.3}$	—
Positive Control Sera				
Human EKC* immune		$10^{-3.5}$	$10^{-6.0}$	2.5
Rabbit hyperimmune to Sanders-Braley EKC virus		$10^{-3.8}$	$10^{-6.5}$	2.7
Rabbit hyperimmune to SLE† virus (Webster strain)		$<10^{-4.0}$	$10^{-6.5}$	>2.5

* EKC—epidemic keratoconjunctivitis.
† SLE—St. Louis encephalitis.

eye-washings and conjunctival scrapings. Uninoculated control-cultures were run simultaneously. In some cases, small cultures in test tubes were inoculated with the washings first; then, 24 hours later, flasks were inoculated with ground tissue and fluid from these tubes.

Subcultures were made once a week, and to test for the presence of virus, 0.03 ml. of ground tissue and culture fluid were inoculated into five or more 14 to 16 gm. male albino mice at each passage by the intracerebral route. Usually half of the control and test animals were injected subcutaneously with 5.0 mg. cortisone acetate (Merck) prior to inoculation with the test material. All mice were kept under observation for one month.

1. *Windsor cases.* Isolations were attempted on eye washings and conjunctival scrapings taken in a small quantity of gelatin-buffered saline, from three of the more severe cases, during the early acute stage of the disease. This material was frozen immediately and kept stored at −60°C. until inoculated into tissue cultures, three or four months later. Ten tissue-culture passages were completed in each of the three series.

At about the time of completion of the three series of cultures from the Windsor cases, a few local cases of epidemic keratoconjunctivitis were observed in the Toronto area. Since these cases provided fresh material, isolations of virus were next attempted from these Toronto cases instead of the Windsor ones.

2. *Toronto cases.* Separate isolations were attempted from eye washings and conjunctival scrapings from five patients (seven eyes). The washings were taken from the infected eyes between three and five days after symptoms appeared, and were inoculated directly into tissue cultures as soon as possible, and without storage in all but one instance. Three of the seven series of tissue cultures were carried to the 15th passage, three to the 10th, and one to the sixth.

RESULTS

1. *Windsor cases.* In no instance were any symptoms of illness observed in the mice.

2. *Toronto cases.* In two of the seven series, four sick mice were found in the groups inoculated from the early tissue-culture passages. They were characterized by a ruffled coat and a humped back. In one series,

one mouse was found sick two days after inoculation with material from tissue-culture passage No. 3. In the other series, two mice were sick eight days after inoculation from passage No. 1; and one mouse was sick three days after inoculation from passage No. 2. In the control groups, no sick mice were found. Each sick mouse was killed and the brain emulsified and passed to five other mice. From one of these groups of five mice, two were found dead on the eighth day, and therefore could not be passed, but none of the other mice developed any signs of illness.

In six of the seven series, paralyzed mice were found in the groups inoculated from the later tissue-culture passages, both from the test and control cultures. The paralyzed mice were characterized by a complete paralysis of the hind legs and occasionally paralysis of one or both of the fore legs.

In all, 19 mice inoculated from the test cultures were found paralyzed, and nine from the controls. They developed paralysis in from nine to 40 days. The brains were passed to other mice successfully and in each series, one mouse passage (usually from the original paralyzed mouse) was continued serially until the LD_{50} ceased to rise (about nine passages) and the incubation period became fixed (four passages).

Thus strains of virus were established from six of the seven attempted isolations. Four of these had an incubation period of nine to 10 days and an LD_{50} of 2.5 to 3.5. The fifth strain had an incubation period of 12 days and an LD_{50} of 1.8. The sixth strain, after the sixth passage, became contaminated, and at that passage had an incubation period of seven days and an LD_{50} of 3.7.

A number of mice were found dead in the groups inoculated from both the early and late tissue-culture passages in four of the seven series. Presumably these would have been classified as either sick or paralyzed had they been found earlier.

The use of cortisone increased the incidence of infection. A significantly larger proportion of the cortisonized than non-cortisonized mice became paralyzed or were found dead, as can be seen from the data presented in Table 2.

The strains of virus were identified as closely related strains of mouse encephalo-

TABLE 2

THE EFFECT OF CORTISONE ON THE PROPORTION OF MICE DEAD OR PARALYZED AFTER INOCULATION WITH TISSUE-CULTURE MATERIAL

Patient	Cortisone	Test Cultures		Percent	Control Cultures		Percent
		No. Mice Inoculated	No. Mice Affected		No. Mice Inoculated	No. Mice Affected	
L.	cort.*	30	1	3	15	0	0
	non-cort.†	149	1	1	91	1	1
R.G. (O.S.)	cort.	65	10	15	30	3	10
	non-cort.	28	2	7	19	0	0
R.G. (O.D.)	cort.	52	9	17	40	7	17
	non-cort.	37	1	3	15	0	0
D.Q.	cort.	80	3	4	60	10	17
	non-cort.	40	0	0	25	0	0
A.S. (O.S.)	cort.	45	4	9	40	0	0
	non-cort.	25	0	0	0	0	0
A.S. (O.D.)	cort.	50	4	8	35	0	0
	non-cort.	30	1	3	0	0	0

* cort.—cortisone acetate (Merck cortone), 5.0 mg. per mouse.
† non-cort.—no cortisone injected.

myelitis. The Theiler TO (Yale) strain of virus was obtained from the American Type Culture Collection. The characteristics of this strain in mice are development of paralysis in seven to 35 days and an LD_{50} of 4.0 (seventh mouse passage). The host range is limited to mice, cotton rats, and chick embryos.

An attempt to immunize the strain of mice of the Connaught Laboratory against this Theiler TO strain was unsuccessful after 16 injections over a period of four months.

A rabbit was hyperimmunized against this strain, but the log neutralization index only reached a value of 1.3 after 17 injections over a period of four months (4.0 ml. of a 10-percent suspension of mouse brain and cord per injection). Three of the five strains of virus gave this same degree of neutralization with the hyperimmune serum, but two failed to give any neutralization.

In investigating the host range of one of the strains (R.G.), it was found that of rabbits, hamsters, white rats, and cotton rats, only cotton rats were susceptible (two died out of nine injected). Another strain (L.) was successfully adapted to the chorioallantoic membrane of the chicken embryo.

Apparently one or more strains of a latent mouse-encephalomyelitis virus had been activated through the use of cortisone, combined with the technique of serial passage of embryonic mouse-brain in tissue culture, and the procedure of keeping the mice under observation for a period of one month after inoculation.

Discussion

The failure to demonstrate neutralizing antibodies against the Sanders-Braley epidemic keratoconjunctivitis virus in serum taken from 38 Windsor and five Toronto cases indicates that the causative virus of epidemic keratoconjunctivitis in Ontario is not related to the Sanders-Braley strain of epidemic keratoconjunctivitis virus. Cheever[4] and Cockburn, et al.,[6] working with the same strain of epidemic keratoconjunctivitis virus, were also unable to find neutralizing antibodies in sera from patients with this disease in the midwestern part of the United States during 1951-53.

Although no isolation of the epidemic keratoconjunctivitis virus was accomplished, a number of mice became sick after inoculation with tissue-culture material from the early passages in two out of 10 attempted isolations. The characteristics of the mice resembled those described by Sanders and Alexander[8] in the original isolation. However, it was not possible to establish any strains of virus from the sick mice by subsequent mouse passage.

Strains of mouse-encephalomyelitis virus were isolated in six out of 10 attempts to isolate the virus of epidemic keratoconjunctivitis. A number of factors probably were important in activating a latent mouse-virus infection, namely the use of cortisone in the mice inoculated with tissue-culture material, the procedure by which mice were kept under observation for one month, and the tissue-culture technique itself, which involved serial passage of embryonic mouse brain.

Sanders, in 1941, ruled out the possibility that he had isolated strains of spontaneous mouse encephalomyelitis virus, by doing what he called cross-infection tests using the Theiler G.D. VII strain. Thus the strain of epidemic keratoconjunctivitis originally isolated by Sanders was not related to Theiler's virus.

Summary

1. Sixty-one sera from patients recovered from epidemic keratoconjunctivitis in the Windsor, Ontario, epidemic in 1951, and eight sera from patients in Toronto, Ontario, failed to show neutralizing antibodies to the Sanders-Braley strain of epidemic keratoconjunctivitis virus.

2. Eight attempts to isolate the virus of epidemic keratoconjunctivitis from eye-washings and conjunctival scrapings using tissue culture methods were made. A number of mice became sick following intracerebral inoculation of tissue-culture material from the early passages. It was not possible

to establish strains of virus by further passage of brains from these sick mice. From the later passages, five strains of virus were established, and these were identified as strains of mouse-encephalomyelitis virus closely related to the Theiler TO strain. The use of cortisone increased the incidence of paralysis and death in the mice inoculated from both the test and control cultures.

Banting Institute,
University of Toronto (5).

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Mrs. Ruth McKneeson participated in the early part of the work, and Miss Irene Miller and Mrs. Violet Simmons gave technical assistance.

The cortisone used in this work was supplied by Merck & Co.

References

1. McGavin, M. F., Boley, J. P., and Ormsby, H. L.: Epidemic keratoconjunctivitis. Canadian M.A.J., **69**:137-140, 1953.
2. Hogan, M. J., and Crawford, J. W.: Epidemic keratoconjunctivitis (superficial punctate keratitis, keratitis subepithelialis, keratitis maculosa, keratitis nummularis): With a review of the literature and a report of 125 cases. Am. J. Ophth., **25**:1059-1078, 1942.
3. Sanders, M., and Alexander, R. C.: Epidemic keratoconjunctivitis: Isolation and identification of a virus. J. Exper. Med., **77**:71-96, 1943.
4. Cheever, F. S.: A possible relationship between viruses of St. Louis encephalitis and epidemic keratoconjunctivitis. Proc. Soc. Exper. Biol. & Med., **77**:125-129, 1951.
5. Parker, R. C.: Methods of Tissue Culture. New York, Hoeber, 1950.
6. Cockburn, T. A., Nitowsky, H., Robinson, T., and Cheever, F. S.: Epidemic keratoconjunctivitis. Am. J. Ophth., **36**:1367-1372, 1953.

TEMPORAL ARTERITIS SYNDROME WITH OCULAR INVOLVEMENT*

A REPORT OF TWO CASES

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Arteritis of the temporal vessels, first reported in 1930 by Schmidt,[1] has been recognized as a clinical entity since its description by Horton, Magath, and Brown[2] in 1934. The disease is essentially an arteritis and periarteritis of the temporal vessels characterized by a hard, cordlike thickening of the temporal arteries, by the presence of easily palpable nodes along these arteries, and by painful areas over the temple and scalp.

Horton, Magath, and Brown,[2] and the writers immediately following them, believed it to be a disease of the temporal arteries only, but later observers, including Bruce[3] and Post and Sanders,[4] found that other vessels of the body could also be affected, and it is now known to have involved the carotid, subclavian, coronary, renal, mesenteric, innominate, iliac, pulmonary, femoral, brachial, and dorsalis pedis arteries.

The original term "temporal arteritis," and the "cranial arteritis" suggested by Curtis,[5] are thus clearly inadequate but, until enough is known about the disease to suggest a more appropriate name, the term "temporal arteritis syndrome" perhaps best serves the purpose.

The disease seems to be limited to elderly patients and to occur in females in some 75 percent of cases (Bruce[3]). The onset is characterized by more or less constant headache,

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Dedicated to Prof. Karl Lindner, with all good wishes and with deep appreciation, on the occasion of his seventieth birthday.

general malaise and lassitude, weakness, slight but often long-continued fever, night sweats, anorexia, loss of weight, anemia, and mild leukocytosis.

The headache is boring in type, is usually more marked on one side, tends to be more severe at night, and may be of the intractable type that is not relieved by barbiturates or morphine. In from two to five weeks after onset, the temporal arteries become very prominent and tortuous and, along their courses appear inflamed, raised nodules, very painful and tender to touch. In the early stages there is a pulsation in the arteries which subsequently disappears. Usually, though not always (Walsh[6]), the patient appears to be ill out of all proportion to the visible signs.

Pathologically the lesion is a chronic periarteritis and arteritis. There is a thickening of the arterial wall involving all three coats and a narrowing and occlusion of the lumen. The intima is hypertrophied, the media necrotic, and both the intima and the adventitia are infiltrated with lymphocytes and polymorphonuclear cells; giant cells are usually present in the media.

Etiology

The etiology of the disease is still unknown. According to Johnson, Harley, and Horton,[7] the microscopic appearance of resected vessels suggests a chronic inflammatory process in most instances and a degenerative process in some instances. The general clinical picture and the usually associated symptoms of malaise, headache, weakness, fever, and mild leukocytosis extending over a long time certainly suggest a chronic infectious process, but a conclusion to this effect is opposed by the fact that the disease is found in females in the majority of cases and that the age group is between 55 and 80 years.

Bessiere and Julien[8] and others have classified the disease with the thrombosing types of periarteritis, such as periarteritis nodosa, thromboangiitis obliterans of Buerger, and rheumatismal arteritis, all of which are included in the group of adaptation diseases described by Hans Selye. Peralta and Gomez Angulo[9] regard the disease as a generalized process with elective localization in the cranial vessels.

Ocular involvement

Although eye findings were described in connection with many of the cases of temporal arteritis reported in the literature, Johnson, Harley, and Horton[7] were the first observers to give them serious consideration.

Subsequently, in the most comprehensive study of ocular involvement that has appeared, Bruce[3] reviewed 84 published cases of temporal arteritis and found ocular involvement in 40 percent of them. Of the patients so involved, 38 percent became totally blind in both eyes, 27 percent became blind in one eye, and seven percent had various degrees of visual impairment.

According to Bergouignan and Julien,[10] the ocular complications can be divided into two general classes: (1) Benign complications consisting of diplopia, muscular paresis or transient ptosis, pain in the eyeballs, and photophobia; and (2) severe complications consisting of thrombosis of the central retinal artery, or, more commonly, of an eschemic optic neuritis with an edematous or pale papilla.

As Wagener[11] points out, the eye itself seems to be attacked in two ways:

First, a closure of the retinal arteries in the nerve may occur. This is usually permanent although in rare instances, when the closure is due to a spasm, there may be some restoration of vision.

Second, the ischemia may take place behind the entry of the retinal vessels into the nerve, and in this event symptoms indistinguishable from an acute retrobulbar neuritis develop.

Occasionally (Meyer-Schwickerath[12]) there may be a combination of the two

fundus complications: obstruction of the central artery and ischemic neuritis. In addition, there is a group of complications that Wagener calls the "indeterminate group" which includes unexplained loss of vision, subretinal hemorrhage, retinal phlebitis, general narrowing of the arteries, and periarterial sheathing.

Bruce[3] found that 18 percent of the ocularly involved cases showed occlusion of the artery, 38 percent had ischemic retrobulbar neuritis, and 44 percent displayed one or another of Wagener's indeterminate group of manifestations.

Diagnosis

In the typical case, diagnosis is relatively easy because of the swollen, tender, painful temporal arteries. These are characteristically palpable and tender during the period of boring headache (Hoyt, Perera, and Kauvar[13]) and should be palpated by the ophthalmologist in all cases of occlusion of the retinal vessels and of acute retrobulbar neuritis.

The ocular symptoms may, however, postdate by several weeks or months the period in which the temporal arteries are most acutely inflamed. In Post's case[14] there was a lapse of three months, in one of the cases reported by Johnson, Harley, and Horton[7] there was a lapse of four months, and in the case reported by Smith and Greene[15] there was a lapse of six weeks.

As pointed out by Anderson,[16] the bilateral pain which is in many cases the early presenting feature is thus not observed in connection with the ocular symptoms and diagnosis may depend upon the history and upon the internist's examination. In the absence of an adequate history some cases are undoubtedly diagnosed as retrobulbar neuritis followed by optic atrophy.

At times, as in the first case reported here, there may be no definite history of temporal artery tenderness but only of the temporal pain connected with the ocular symptoms, and if the involvement is limited to the ophthalmic artery at the time of examination there may still be no temporal artery tenderness.

Treatment

It is rather generally agreed that treatment is ineffective. Bergouignan,[17] Harrington,[18] and others have reported that sectioning of the temporal artery gives relief from pain, especially if performed where the artery shows thrombosis. Harrington had two cases that were relieved of all pain by such a resection.

Roberts and Askey[19] succeeded in controlling the pain by injecting procaine around the affected vessels.

Bruce[3] stated that vasodilators were useless but Anderson[16] felt this to be unwarrantably pessimistic; he felt that since a percentage of cases were certainly due to vasospasm, the percentage of cases showing recovery would be higher if vasodilators were used, possibly with a sympathetic block induced by means of anticoagulant therapy.

Duggan[20] reported the successful use of vasodilators in a case in which the patient had lost the sight of one eye; when it began to fail in the second eye the use of niacin halted the process and restored the vision in this eye completely.

Cuppers[21] reports the unsuccessful treatment of a case with retrobulbar injections of priscol, penicillin, capusal, betaxin, and pyramidon; in spite of treatment bilateral optic atrophy with amaurosis developed within two months.

Case reports

Case 1

T. K. (U.C. 143206), a 55-year-old woman, entered the University of California Clinic on February 29, 1952, complaining of severe pain in and about the left eye of four months' duration and accompanied by a boring headache. She had not felt very well during this period but there was no history

of severe malaise, loss of weight, and so forth.

The pain began in the left temple, where it was very severe, and radiated into the left eye. It was intermittent and aggravated by motion of the left eye. Three months after the onset of the pain she had noted a diminution of vision. This was slowly progressive until at the time of entry she was just able to distinguish light.

The patient stated that prior to the onset of the present complaint she had had occasional episodes of sharp pain in the left side of her face associated with lacrimation and paresthesia on the same side. Medical examinations conducted in 1948 were essentially negative.

Physical examination. The patient was a well-nourished woman 55 years of age. Temperature was 98.8, pulse 80, respiration 20, blood pressure 140/85 mm. Hg. With the exception of the findings of the eye examination to be described, the physical examination was essentially negative. Laboratory studies, including a uveitis survey, were negative. Biopsy of the left temporal artery showed some generalized contraction but no signs of inflammation of the vessel.

Eye examination. Vision was R.E., 20/20 (with correction); L.E., bare light perception.

External eye examination. Negative except for definite tenderness of left globe on even light pressure; no evidence of involvement of the ocular muscles. Fundi: R.E., media clear; mild changes of hypertension; fundus otherwise essentially negative. L.E., media clear; edema of the disc of 2.5D. with venous engorgement; artery showed some arteritis and partial occlusion; edema and ischemia of the retina; some exudate on the disc and flame-shaped hemorrhages adjacent to the disc (fig. 1).

Intraocular pressure was, R.E., 18.5 mm. Hg; L.E., 16 mm. Hg (Schiøtz). Exophthalmometer reading (bar at 97): R.E., 16 mm.; L.E., 16 mm.

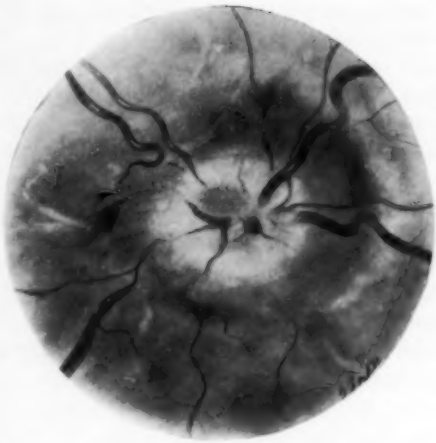

Fig. 1 (Cordes). *Case 1.* Fundus of left eye. (Drawn from fundus photograph.)

Diagnosis. Temporal arteritis syndrome involving left ophthalmic artery.

Two months after the initial examination there was complete optic atrophy of the left disc with marked attenuation and sheathing of the arteries. The right eye remained unchanged.

CASE 2

Caroline B. (U.C. 188071), a 69-year-old woman, entered the University Hospital on February 10, 1949, complaining of severe pain in the temporal region associated with sudden loss of vision in the right eye. On four occasions before entry she had had severe pains in the right temple, jaw, and palate. These pains were at first mild, infrequent, and transient, but had of late become constant and associated with continuous throbbing of the head. She had apparently precipitated severe pains on several occasions by chewing her food.

In the early part of January she had entered the hospital in Oakland for study because of the pain and tenderness of the temporal region, especially over the arteries. Studies at this time failed to reveal the cause of her difficulties although her internist con-

sidered dermatomyositis, periarteritis nodosa, and scleroderma as possibilities. The final diagnosis at that time was "generalized arteriosclerosis and osteoarthritis."

Four days before entry the patient had a sudden loss of vision in the right eye accompanied by moderately severe pain and a boring headache that had been present for a number of days and had been controlled with difficulty.

Physical examination. The patient was a 69-year-old woman in pain. Temperature 99, respiration 85, blood pressure 155/80 mm. Hg. The general physical examination was negative except for a small ventral hernia, arteriosclerosis, and osteoarthritis. X-ray studies of the skull were negative and a carotid arteriogram on the right failed to demonstrate any evidence of disease. Dental X-ray studies revealed two abscessed teeth. There was also chronic sinus disease. The sedimentation rate was elevated but other laboratory findings were essentially negative.

Eye examination. Vision was R.E., bare light perception., L.E., 20/20 (with correction).

External eye examination. Negative; no evidence of involvement of the extraocular muscles. Fundi: R.E., the pupil was middilated and reacted very slowly to light with a minimal hippus type response. The disc was edematous and elevated two diopters with the edema extending into the surrounding retina about two disc diameters. The disc was pale. There were no hemorrhages. The retinal arteries seemed rather small but aside from this were within normal limits. The light field was limited to a small area temporally and above. L.E., essentially negative.

Intraocular pressure was: R.E. 22 mm. Hg; L.E. 20 mm. Hg (Schiøtz). Exophthalmometer reading (bar 100): R.E., 18; L.E., 19.

Diagnosis. Temporal arteritis syndrome involving right ophthalmic artery with possible previous temporal artery involvement.

The patient returned to her oculist, Dr. Owen Dickson, to whom I am indebted for the following information:

March 4, 1949, (three weeks after the first examination). The edema was less but there was a hemorrhage at the disc margin. At this time the two involved teeth were removed and the patient experienced marked relief from pain.

March 14, 1949. The edema was much less but there was definite evidence of atrophy and of a narrowing of the arteries.

June 6, 1949. There was complete atrophy of the right disc with attenuation of the arteries. The vision of the left eye remained unchanged.

Summary

The literature on the temporal arteritis syndrome, with special reference to the ocular complications, is reviewed, and two cases are reported in which there was involvement of the ophthalmic artery.

The first case was that of a 55-year-old white woman with a history of loss of vision in the left eye, severe pain in and about the left eye, and a boring headache. Examination of the left eye revealed arteritis and partial occlusion of the retinal artery. The loss of vision had not started until three months after the onset of the left-sided pain. Biopsy of the left temporal artery showed some contraction but no inflammation. The patient did not display the typical symptoms of general malaise and fever. No contributing causes were found in the general physical examination. Two months after the initial examination there was complete optic atrophy of the left disc with marked attenuation and sheathing of the arteries.

In the second case, that of a 69-year-old white woman, a sudden loss of vision developed in the right eye accompanied by severe pain and headache. Five weeks previously she had had what appeared to be a typical temporal arteritis. The right eye revealed an ischemic optic neuritis. This was followed four months later by complete optic atrophy with attenuation of the arteries. In

addition to generalized arteriosclerosis and osteoarthritis, the general physical examination revealed two abscessed teeth and chronic sinus disease. Considerable relief from pain followed removal of the two abscessed teeth.

Thus in the first of the two cases, we have an example of the type in which there is an occlusion of the central retinal artery with arteritis; while in the second patient there was an ischemic type of optic neuritis. Both of these patients developed the ocular signs and symptoms after all evidence of temporal arteritis had subsided—three months later in the first case and five weeks after the temporal arteritis in the second case.

Since this arterial disease is not limited to the temporal and ophthalmic arteries as originally thought but is found scattered throughout the vessels of the body, the term "temporal arteritis syndrome" does not properly describe it but can serve the purpose until enough more is known about the disease to suggest a better name.

384 Post Street (8).

References

1. Schmidt, M.: Intracranial aneurysms. Brain, **53**:489 (Jan.) 1931.
2. Horton, B. T., Magath, T. B., and Brown, G. E.: Arteritis of the temporal vessels: A previously undescribed form. Internal Med., **53**:400-409 (Mar.) 1934.
3. Bruce, G. M.: Temporal arteritis as a cause of blindness: Review of literature and report of a case. Tr. Am. Ophth. Soc., **47**:300-316, 1949.
4. Post, L. T., and Sanders, T. E.: Temporal arteritis. Am. J. Ophth., **27**:19-25 (Jan.) 1944.
5. Curtis, H. C.: Cranial arteritis: A review of the literature with report of a case with total blindness. Am. J. M. Sc., **212**:437-446 (Oct.) 1946.
6. Walsh, F. B.: Clinical Neuro-ophthalmology, Baltimore, Williams & Wilkins, 1947, p. 1327.
7. Johnson, R. H., Harley, R. H., and Horton, B. T.: Arteritis of the temporal vessels with loss of vision. Am. J. Ophth., **26**:147-150 (Feb.) 1948.
8. Bessiere, E., and Julien, R. G.: Lesions of the optic disc in the syndrome of temporal arteritis: Frequency of ischemic edema of the papilla. Arch. d'ophtal., **10**:701-714, 1951.
9. Peralta, G., and Gomez Angulo, J.: J.M. Hospital de Santa Maria Magdalena, Almeria. Arteritis de la temporal. Actualidad Medica (Granada), **24**:287, 646-649, 1948.
10. Bergouignan, M., and Julien, R.: Constations ophtalmoscopiques dans deux cas d'arterite temporale. Rev. oto-neuro-oftal., **22**:540-545, 1950.
11. Wagener, H. P.: Temporal arteritis with loss of vision. Am. J. M. Sc., **212**:225-228 (Aug.) 1946.
12. Meyer-Schwickerath, G.: Univ.-Augenklin., Hamburg-Eppendorf, Erblindung bei Arteritis temporalis. Arztl. Wchnschr., **6**:704-707, 1951.
13. Hoyt, L. H., Perera, C. A., and Kauvar, A. J.: Temporal arteritis. New England J. Med., **225**:1001-1004, 1941.
14. Post, L. T.: Discussion of Bruce's paper. Tr. Am. Ophth. Soc., **47**:313, 1949.
15. Smith, C., and Greene, P. B.: Ocular complications of temporal arteritis. Am. J. Ophth., **32**:685-689 (May) 1949.
16. Anderson, B.: Discussion of Bruce's paper. Tr. Am. Ophth. Soc., **47**:310, 1949.
17. Bergouignan, M.: Apropos d'un troisieme cas d'arterite temporale. Rev. oto-neuro-oftal., **22**:545-546, 1950.
18. Harrington, D. O.: Personal communication.
19. Roberts, A. M., and Askey, J. M.: Temporal arteritis. Relief of headache by injection of procaine hydrochloride. J.A.M.A., **137**:697-699 (June) 1948.
20. Duggan, W. F.: Discussion of Bruce's paper. Tr. Am. Ophth. Soc., **47**:315, 1949.
21. Cuppers, C.: Univ. Klin. für Augenkrankh. Frankfurt a M. über die Mitbeteiligung des Auges bei der Arteritis temporalis. Klin. Monatsbl. f. Augenh., **118**:645-647, 1951.

SUBARACHNOID HEMORRHAGE

Intraocular symptoms and their pathogenesis

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The only means to avoid the many misunderstandings regarding the frequency and the pathogenesis of intraocular signs in subarachnoid hemorrhage is a discussion of the subject based on personal investigations, the clinical as well as the anatomic data. This article is based on:

1. A review of the clinical records of 225 patients with a spontaneous subarachnoid hemorrhage, all observed in the same neurologic department. All the patients had undergone one or more ophthalmologic examinations.

2. A survey of serial sections of the globes and the orbital part of the optic nerves of three patients with a spontaneous subarachnoid hemorrhage, who showed intraocular hemorrhages. This investigation was intended to establish the mechanism by which these intraocular hemorrhages occur.

3. A survey of serial sections of the bony optic canal and its surrounding tissues in three patients with spontaneous subarachnoid hemorrhage, who showed at the postmortem examination a hemorrhage between the optic nerve sheaths. These patients had not shown intraocular hemorrhages during their life. This investigation was intended to prove the presence or absence of an open communication between the cerebral subarachnoid space and the subarachnoid space around the optic nerves.

4. Experiments in which a mixture of gelatin and India ink was injected suboccipitally into the subarachnoid space of human cadavers to prove in a different way the presence or absence of the open communication mentioned under (3).

Clinical intraocular symptoms of spontaneous subarachnoid hemorrhage

The Amsterdam University Neurological Department (present chief: Prof. A. Biemond, M.D.) has been profoundly interested during more than 20 years in the clinical and anatomic data of spontaneous subarachnoid hemorrhage.

The first study on the subject by Biemond and Ter Braak[1] appeared in 1933. This study concerned 40 patients and the authors entered at great length into the ophthalmologic symptoms.

In 1944, I published an article,[2] partly based on a survey of the clinical records of another 75 patients. A paper read in 1950 before the International Ophthalmological Congress in London[3] was partly based on another series of 46 cases, and recent studies, closed November 1, 1953, concern another 64 cases. Thus I am able to report on 225 patients who all had been subjected to a careful clinical observation in the same neurologic department, including one or more ophthalmologic examinations.

The clinical records of these 225 patients reveal that only 45 patients showed intraocular hemorrhages and only 38 patients showed papilledema. Thus *only one fifth of all patients with a spontaneous subarachnoid hemorrhage show intraocular hemorrhages and only one sixth of all patients show papilledema!*

These findings contrast with many statements in the literature. I only cite Duke-Elder[4]: "papilloedema is the most constant symptom" and "retinal haemorrhages are associated with the majority of cases." Walsh and Hedges[5] in their authoritative paper, stated that retinal subhyaloid hemorrhages occurred in 33 percent of their 90 cases of spontaneous subarachnoid hemorrhage. These figures, however, concern only cases of optic-nerve sheath hemorrhages observed in a pathology department and have little clinical value.

The retinal hemorrhages can occur within,

in front of, and behind the retina. When they are subhyaloid they may be very large.

Cordes[6] recently published a beautiful picture of such an extensive subhyaloid hemorrhage. Often, however, only small hemorrhages are found; sometimes they are unilateral, sometimes bilateral. Riddoch and Goulden[7] and Walsh[8] stated that the retinal hemorrhages are usually small. I also must emphasize that the presence of only small retinal hemorrhages is in no way a counter-argument against the diagnosis of a spontaneous subarachnoid hemorrhage.

The almost pathognomonic picture of large profuse hemorrhages, situated in the neighborhood of the papilla, is seldom encountered. The study of the clinical and anatomic records revealed that, as a rule, the intracranial hemorrhage is more profuse in cases of bilateral retinal hemorrhage than in cases of unilateral hemorrhage.

Retinal hemorrhages are prognostically unfavorable: more than 50 percent of the patients with retinal hemorrhages have died against less than 25 percent of the patients without retinal hemorrhages. The same conclusion can be drawn from the figures of Walsh and Hedges. The retinal hemorrhages can be found within one hour after the onset of the intracranical hemorrhage.

The *papilledema* in most cases is marked by its moderate degree. Time and again the papilla was described as being "blurred" or "lightly swollen." More pronounced papilledema was, except for one patient, always accompanied by retinal hemorrhages. A choked disc can present serious diagnostic difficulties; especially when no retinal hemorrhages are present. Biemont and Ter Braak gave some examples of it; Tureen[9] emphasized it as well.

Pathogenesis of the intraocular symptoms

An anatomic study in serial sections of the globes and the orbital part of the optic nerves in three cases of spontaneous subarachnoid hemorrhage with intraocular bilateral hemorrhages, described in 1944,[2] revealed that the retinal hemorrhages were due to ruptured retinal veins. Pictures of ruptured retinal veins were given. Later Walsh and Hedges showed similiar pictures. The intraocular hemorrhage did not communicate via the lamina cribrosa or the sclera with the hemorrhage within the optic-nerve sheaths. It was concluded that the venous ruptures were due to an acute overfilling of the branches of the central retinal vein caused by:

1. The sudden closure of the stem of the central retinal vein where it traverses the intervaginal spaces around the optic nerve.
2. A back pressure of venous blood from the stem of the vein into the retinal branches, due to a compression of the optic nerve itself.
3. The circumstance that the supply of arterial blood to the retina is not impeded by the high pressure within the intervaginal spaces. The open communication between the subarachnoid space of the brain and the subarachnoid space around the optic nerves was at that time accepted as a proven fact that did not need further discussion.

Ballantyne, however, had published a year before an article on the subject[10] that had not reached the German-occupied Netherlands. Ballantyne had stated:

"It is now generally agreed that the blood in the cerebral subarachnoid space does not, as a rule, travel through the optic foramen into the subarachnoid space of the optic nerve."

He had found hemorrhages among all tissues of the orbit and surrounding the vessels of the chiasm and optic tracts, and thought that this "can only be explained by a sudden rise of intracranial pressure causing a stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit."

This view was afterward supported by Walsh and Hedges. They felt established beyond question "that the presence of increased venous pressure is transmitted from

the cranial cavity to the orbital veins. The degree and extent of venous ruptures are dependent on the rapidity of onset and the magnitude of elevation of venous pressure in the brain and orbit." Recently Cordes also accepted this view of Ballantyne and Walsh and Hedges.

It is easy to demonstrate, however, that this view is incorrect. The veins in the orbit have many anastomoses with the facial vein, namely, the supraorbital, the angular, and the deep facial veins, and the pterygoid plexus (see figure 249 of the third edition of *The Anatomy of the Eye and Orbit* by Eugene Wolff[11]).

It is clear that a sudden rise of intracranial pressure never can produce a stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit, as the venous blood finds an easy outlet into the facial vein through these anastomoses. Only an obliteration of the internal jugular vein could be capable of producing a venous congestion as is presumed by Ballantyne, and Walsh and Hedges.

During the London Congress in 1950, I could demonstrate pictures of serial longitudinal and cross sections of the optic nerves from the pituitary up to the eye in three cases of spontaneous subarachnoid hemorrhage. These photographs proved beyond question that an open communication exists between the subarachnoid space of the brain and that of the optic nerves in the orbit.

It was apparent from our investigation that the optic nerve receives its arachnoid sheath anterior to the chiasm. The arachnoid space narrows considerably in the optic canal and is widest lateral to the nerve, narrower medially, still narrower inferiorly, and narrowest superiorly. Above the nerve, the space consists only of some layers of endothelial cells and is traversed by connective-tissue bundles which attach the nerve to the dura. The space was clearly marked by erythrocytes and its presence all around the nerve in the optic canal was evident.

Vail[12] has rightly reminded that cerebrospinal fluid is contained between two layers of the arachnoid, one of which is adherent to the dura and the other to the pia.

The passage of blood through the open communication can happen with such a force that intradural hemorrhages occur, which originate from the subdural space, as is proven by the ruptured innermost layers of the dura. In one of the cases (described in 1944[2]) the central retinal vein was almost torn to pieces due to the acute widening of the intervaginal spaces. A venous hemorrhage never would develop such a destroying force.

Riddoch and Goulden have also pointed to the fact that the walls of the central retinal vein in the intervaginal spaces will not only be compressed but will also be drawn together, as the optic-nerve sheaths will be widely drawn apart by their sudden filling with blood.

Moreover, I demonstrated in London the presence of an open communication through the optic canal in a quite different way. With the aid of a neurosurgeon, a mixture of gelatin (five percent) and India ink was injected suboccipitally into the subarachnoid space of human cadavers. The cadavers were left face down after the injection. The next morning the optic canal and the optic nerve were cut out, and in some of the serial sections the India ink could be traced through the whole length of the subarachnoid space in the optic canal.

It is very remarkable that Walsh and Hedges report similiar injection experiments on human cadavers and publish four convincing photographs which prove that the India ink has penetrated from the cerebral subarachnoid space through the optic canal into the arachnoid space of the optic nerve in the orbit. Their Figures 8, 10, and 11 clearly demonstrate that the dye has not penetrated into the subdural space. In their conclusion, however, they did not mention a single word concerning these experiments which did not correspond with Ballantyne's view.

Our investigations and those of Walsh and Hedges conclusively prove that an open communication is present between the subarachnoid space of the brain and the subarachnoid space of the optic nerve.

On the basis of these studies the conclusion may be drawn that a spontaneous subarachnoid hemorrhage causes a sudden intracranial hypertension and that the blood or the cerebral liquor is forced under high pressure through this open communication into the subarachnoid space of the optic nerves.

The central retinal vein, where it traverses the intervaginal spaces, and its choroidal anastomoses near the lamina cribrosa (Miller and Cuttino[13]) are closed by this high pressure. At the same time a back pressure of venous blood from the stem of the central retinal vein into its retinal branches occurs by a compression of the nerve itself. As the central retinal artery will supply even more blood than usual to the retina (see below), the retinal veins and capillaries are unable to absorb this blood and ruptures will occur.

Some points still need some discussion.

First of all, the fact that Ballantyne, as well as Walsh and Hedges, found hemorrhages surrounding the vessels of the chiasm and optic tracts, the intracanalicular portion of the optic nerve, the orbital dura, and all the orbital tissues. According to these authors these hemorrhages could only be explained by a stasis in the venous channels.

Bruckner, et al.,[14] however, stressed the fact that the ophthalmic artery is the only branch of the internal carotid which, immediately behind its intracranial origin, supplies an extracranial area. In contrast to that of all other branches of the internal carotid, this area is not submitted to intracranial hypertension, and therefore more blood than is usual passes along the ophthalmic artery after a spontaneous subarachnoid hemorrhage. The acute plethora of the ophthalmic artery spreads to the central retinal artery and its other branches in the orbit. As small recurrent branches pass back through the superior orbital fissure or return through the dural sheath of the optic nerve, the hemorrhages throughout the whole orbit, and even the multiple hemorrhages behind the optic foramen, can be more satisfactorily explained by arterial overfilling than by a venous stasis.

A second fact that remains to be explained is the relative infrequency of the occurrence of intraocular symptoms in patients with a spontaneous subarachnoid hemorrhage. The skull is a closed space with little outlet, and filled with pulsating brain tissue and a little liquor. The subarachnoid outlet through the optic canals is funnel-shaped (see the photographs in the *Acta* of the London Congress).

It can be readily appreciated that these funnels become partly or totally tampooned by the acute overfilling with blood. Moreover, we know that the blood from many ruptured aneurysms only oozes into the subarachnoid space, owing to the presence of organized thrombus in the aneurysm, or the presence of arachnoidal adhesions in the basal cisternae from previous hemorrhages. In these cases the blood may be "massaged" gradually through the optic canal by the cerebral pulsations without producing a marked hypertension in the intervaginal spaces of the optic nerves.

Summary

A review of 225 clinical reports of patients with a spontaneous subarachnoid hemorrhage revealed that only one fifth of the patients showed retinal hemorrhages, and only one sixth showed papilledema.

The occurrence of intraocular hemorrhages in patients with spontaneous subarachnoid hemorrhage is explained by a sudden intracranial hypertension that forces blood and (or) liquor under high pressure into the subarachnoid space of the optic nerve through an open communication in the optic canal.

Compression of the central retinal vein where it traverses the intervaginal spaces

and compression of the choroidal anastomoses of the central retinal vein cause a venous stasis.

The congestion is aggravated by increased blood flow through the central retinal artery. These forces are sufficient to cause rupture of retinal veins and capillaries in peracute cases.

Nieuwe Binnenweg 157.

REFERENCES

1. Biemond, A., and Ter Braak, J. W.: Ueber die sogenannten spontanen subarachnoidalen Blutungen und ihre Beziehung zum Aneurysma der Hirngefäsze. Deutsche. Ztschr. Nervenh., **132**:4, 1933.
2. Manschot, W. A.: The fundus oculi in subarachnoid haemorrhage. Acta Ophth., **22**:281, 1944.
3. Manschot, W. A., and Hampe, J. F.: The origin of ocular symptoms in spontaneous subarachnoid haemorrhages. Acta XVI. Cong. Ophth. (London), 1950, v. I, p. 356.
4. Duke-Elder, W. S.: Textbook of Ophthalmology. London, Kimpton, 1941, v. 3, p. 2939.
5. Walsh, F. B., and Hedges, T. R.: Optic nerve sheath hemorrhage. Am. J. Ophth., **34**:509, 1951.
6. Cordes, F. C.: Subhyaloid hemorrhage following subarachnoid hemorrhage. Am. J. Ophth., **36**:1192, 1953.
7. Riddoch, G., and Goulden, C.: On the relationship between subarachnoid and intraocular haemorrhages. Brit. J. Ophth., **9**:209, 1925.
8. Walsh, F. B.: Clinical Neuro-ophthalmology. Baltimore, Williams & Wilkins, 1947, p. 1012.
9. Tureen, L. L.: Lesions of the fundus associated with brain hemorrhage. Arch. Neurol. & Psychiat., **42**:664, 1939.
10. Ballantyne, A. J.: The ocular manifestations of spontaneous subarachnoid haemorrhage. Brit. J. Ophth., **27**:384, 1943.
11. Wolff, E.: The Anatomy of the Eye and Orbit. London, Lewis, 1948, p. 335.
12. Vail, D.: The blood supply of the optic nerve and its clinical significance. Am. J. Ophth., **31**:1, 1948.
13. Miller, A. J., and Cuttino, J. T.: On the mechanism of production of massive preretinal hemorrhages following rupture of a congenital medial-defect intracranial aneurysm. Am. J. Ophth., **31**:15, 1948.
14. Brückner, R, et al.: Zur Genese der Retina- und Opticus-Scheidenblutungen bei akuter intrakranieller Drucksteigerung. Ophthalmologica, **118**:607, 1949.

THE STELLATE FIGURE IN THE MACULAR REGION*

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Twenty-five years ago an eminent pathologist saw some of my earliest photographs of macular stars and said that it was difficult to correlate the fundus changes with the then popular concept that they were in Henle's layer.

Several years later, some short textbook statements referred to stellate retinitis, and assertions were made that when the partial star was on the disc side of the macula it was characteristic of papilledema.

In the preparation of this clinical demonstration many photographs were examined. One of the facts which soon became evident was that the stellate figure was often absent in cases with gross retinal changes, while in others with similar patterns they were present. This was especially noteworthy in patients with papilledema, hypertension, or nephritis.

The presentation will show a wide variety of concomitant fundus changes and special attention will be paid to the size and location of the individual radiations.

The appearance of the macular area may be deceptive, for many stars are so transitory that the examination must be fortunately timed; otherwise they may not be seen. All stars are constantly changing, some almost imperceptibly over a period of weeks, while others pass through their entire cycle in a few days. There may be many spokes in the wheel of exudate, or only one. The visible

* Presented at the 89th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1953.

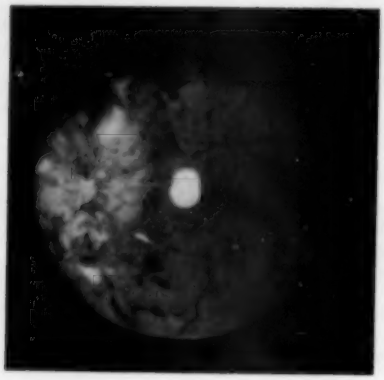

Fig. 1 (Bedell). Macular folds and exudate lines in papilledema.

lines may be so thin and so narrow that the differentiation between puckered retinal folds and faint, gray, broken rays is almost impossible without stereoscopic examinations, preferably of photographs.

The site of the macular figure is usually close to the margin of the fovea and seldom extends over it. The most numerous, as well as the longest and widest, radiations are between the macula and the disc. Even when the circle is complete, the disc side is usually the most prominent, although occasionally very thick rays are below and, still more rarely, above the fovea.

The method of dispersal varies from the progressive decrease of each spoke to the widespread dissemination into thick spots or small, separate dots. All stars tend to resolve but, unfortunately, death frequently supervenes before they are entirely absorbed.

Review of cases

The earliest pathologic alterations in the macula related to the subject are narrow, radiating, dull gray wrinkles as observed in the left fundus of a 17-year-old boy with bilateral papilledema of high degree. There were no puckers in the opposite eye.

The left eye of a 30-year-old man with a right temporoparietal glioma and a marked papilledema, hemorrhages, and peripapillary exudates, showed slightly elevated, gray lines extending from the macula toward the disc.

In the right eye the papilledema was the same but there were no macular radiations.

A 30-year-old man with a hemangioma-blastoma of the cerebellum, left hemisphere, and anterior half of the vermis had 20/20 vision in his right eye without macular involvement (fig. 1.)

The left-eye vision was 20/70. There were a great swelling of the disc, many white exudates, distended veins, narrow arteries with a dark macula, and many radiating, slightly elevated folds and a few short exudate lines. The papilledema was the same in the right eye but there was no suggestion of a star.

One phase of the macular exudate in angiomatosis retinae is well illustrated in the fundus photograph of the right eye of an 11-year-old girl (fig. 2). All of the retinal veins were widely distended, but those in the lower half which went to the peripheral aneurysm were much more distended. The fovea was almost completely concealed by a large mass of exudates radiating from the macula.

Delicate, narrow, linear radiations are occasionally seen in the acute traumatic macular edema described by Berlin.

A 12-year-old boy was photographed 36 hours after he had been struck in his right eye with a stone from a slingshot. The vision was 3/200. There was a hyphema with an

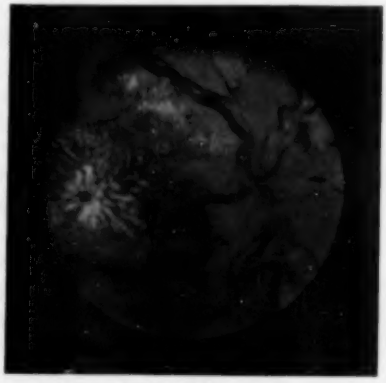

Fig. 2 (Bedell). Complete macular star in angiomatosis retinae.

edematous elevation of the macular region with many fine exudate lines, which were especially marked near the upper and lower margins.

Three days later, the vision was 20/20. The retinal edema was resolved and the lines had disappeared.

A partial star was found in a 62-year-old woman who had blood pressure of 220/90 mm. Hg with marked retinal artery caliber changes. The edema of the retina was especially marked near the inferior margin of the disc and several bright, long, narrow, gray-white, and more or less nodular streaks extended from the disc side of the macula.

One month later, several of the lines had disappeared and those that remained were short and dashlike.

She continued to live for seven years.

A similar nasal-side fan was presented with a low-grade optic neuritis in a woman, aged 37 years, who had been taking reducing pills. The vision had been blurred for six weeks and was accompanied by ocular pain. All physical and laboratory tests were negative.

In the right eye the vision was 20/100. The disc was pale and elevated with moderate retinal edema. There were eight short, oblique lines consisting of small, white dashes on the disc side of the macula.

Two weeks later, there was an increase in the number of the exudate spots but they were not radially arranged.

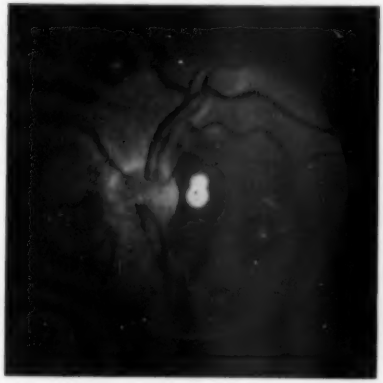

Fig. 3 (Bedell). Optic neuritis with stellate figure.

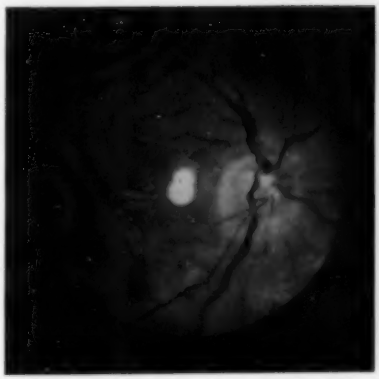

Fig. 4 (Bedell). Disc-side fan of exudate in high NPN.

One month later, the disc was white, the edema much decreased, and the exudates decidedly fewer and smaller, almost pinpoint dots.

Another case of optic neuritis was in a 57-year-old man with left-eye vision of 20/40. The disc was edematous; several thick dot-and-dash lines extended toward it (fig. 3). The systolic blood pressure was 155 mm. Hg and the NPN 21.

The other eye was not involved.

A prominent phase in the development of the macular star is a partial fan extending from the macula toward the disc. This was found in a 46-year-old man who had noted reduced sight for 12 days. The right eye vision was 1/200. There were eight soft-gray, yellow radiations about one disc diameter in length with a tendency to form short rods or broken lines about the macula with a low degree of retinal edema and changes in the caliber of the inferior nasal artery. All physical and clinical tests were negative, except the NPN, which was 54.5 (fig. 4).

Two days later, the lines were thinner and more of them were broken into rods.

At the end of 10 weeks only a few scattered dots remained, the retinal edema had disappeared, and the artery was normal. Vision was 20/30 and the NPN 30.

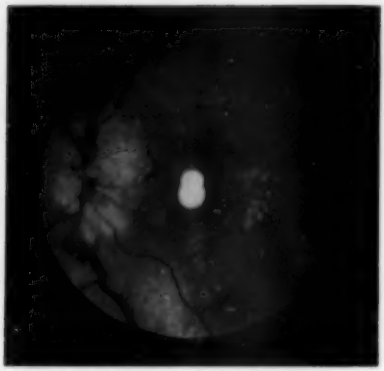

Fig. 5 (Bedell). Macular star in optic neuritis.

He has had no recurrence of retinal or kidney dysfunction. Blood pressure is 140/92 mm. Hg.

A complete macular star consisting of small, white specks may form a circle about the fovea.

This was well shown (fig. 5) in the left eye of a 56-year-old man whose sight had been failing for three weeks. His systolic blood pressure was 120 mm. Hg; NPN 43.6.

There was an elevated, soft-appearing, yellow-white disc with a complete loss of outline. The edema of the retina extended some distance beyond it, the veins were full, the arteries narrow and uneven. There were several deep, small, yellowish exudates to the temporal side of the disc.

Seven weeks later, the disc was well outlined and the retinal edema markedly reduced. Numerous exudates were below the nervehead and in a wide vertical area between it and the macula. The macular radiations were much shorter but those over the fovea were slightly increased in size. The vision was 1/400.

A complete, very large, bright macular star was found in the right eye of a 78-year-old woman. The vision was 6/200. Her systolic blood pressure was 240 mm. Hg. She died shortly afterward.

A 43-year-old disabled veteran with a systolic blood pressure of 190 mm. Hg over a high diastolic had a severe neuroretinal edema, narrow arteries, and a complete ring of exudate about the macula with many long, wide rays covering an area of almost 1.5 disc diameters, largest to the nasal side (fig. 6).

Four months later the retinal edema was decidedly less, the artery changes were more evident, and the macular star partially absorbed into white dashes. He lived for six years.

A woman, aged 66 years, who died a few days later in uremia had unusually thick, white, and long rays with a complete macular star and extreme retinal edema, advanced vein and artery changes.

Diverging lines of white exudate in association with numerous, widely disseminated white spots, small hemorrhages, and minute aneurysms are not infrequent in one phase of diabetic retinopathy.

A 43-year-old woman had had diabetes for seven years and, although her blood sugar was 180, the diet had not been restricted nor was insulin administered. The exudates were scattered about the fundus and also radiated from the macula.

A 33-year-old woman had her pregnancy terminated at four and a half months because on the two preceding days she complained of blurred vision.

Thirty days afterward, her systolic blood pressure was 240 mm. Hg. The vision of the left eye was 20/50 and there was an extreme edema of the retina with scattered, soft, indefinitely outlined gray spots of exudate.

Five weeks later, there was a complete classic star with distinct bright, almost white rays, the longest in the usual position, toward the disc (fig. 7).

Nine months later, the stellate figure was broken into innumerable, bright exudates filling an area almost three disc diameters in size. The arteries were very narrow and uneven in caliber. The edema of the retina was less, but there was one soft, cotton-wool exudate between the inferior temporal vein and artery.

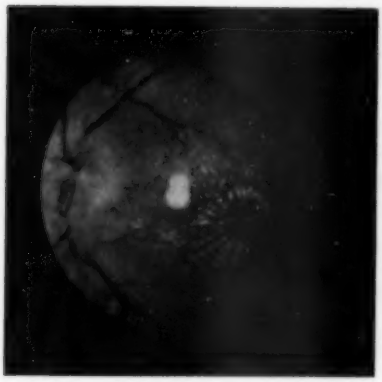

Fig. 6 (Bedell). A large ring with radiations in neuroretinal edema.

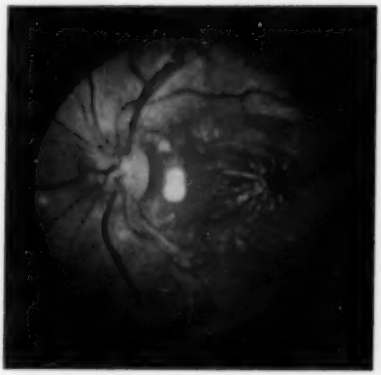

Fig. 7 (Bedell). Complete macular star following premature delivery caused by hypertension.

Three years later she had a bilateral sympathectomy. She is still living, seven years after abortion, although her blood pressure is 238/140 mm. Hg.

A 59-year-old man had had several "heart attacks." He had a right-sided hemianopsia. His systolic blood pressure was 145 mm. Hg when he presented with a long and short line of exudate extending obliquely upward from the macula. These disappeared within five days and he remained comfortable for four years, after which he died suddenly of a heart attack.

A woman, aged 55 years, whose vision with correction was 20/20, had a visible spasm in the superior temporal artery a short distance from the disc. The systolic pressure was 160 mm. Hg.

Six months later, she reported that her blood pressure was 232 mm. Hg and that she had +1 albumin, a fulminating hypertension. There were widespread retinal edema, increased artery caliber changes, superficial hemorrhages, many soft exudates, and four yellowish-gray lines spread from the macula toward the disc. She died two months later.

A 45-year-old woman had blood pressure of 220/124 mm. Hg. In the right eye there was an almost complete macular star (fig. 8); the longest and most distinct radiations were toward the disc side. There were moderate retinal edema, spastic arteries, cotton-wool exudates, and hemorrhages.

In the left eye the changes of retinal edema, narrow arteries, cotton-wool exudates, hemorrhages, and fine exudates were evidence that the star had not as yet formed.

The value of ophthalmoscopic examinations is evidenced in the case of a 31-year-old man who reported that three years previously he had been told that he had nephritis.

Examination of the left eye showed widespread, soft exudates, superficial, flame-shaped hemorrhages, and a low-grade retinal edema. NPN 55.2.

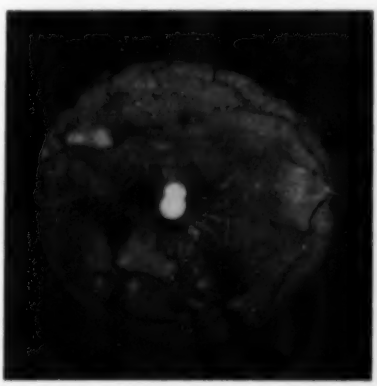

Fig. 8 (Bedell). Hypertensive retinopathy with an almost total star.

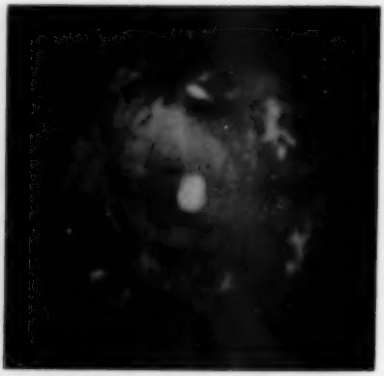

Fig. 9 (Bedell). An extreme nephritic neuroretinopathy with star.

Three months later, there was a considerable decrease in the number and size of the soft exudates. There were no hemorrhages. The artery changes remained the same. In and about the macular region, especially on the disc side, there were several fine exudates, indicating that between visits he had had a macular star.

He died six months later.

In the left eye of a 34-year-old man there was a pale edematous, almost white disc with complete loss of outline. The veins were dilated, the arteries were narrow and of uneven caliber, with soft exudates and several hemorrhages. From the upper half of the macula several lines of exudate radiated. The patient had a similar condition in the right eye. The blood pressure was 230/140 mm. Hg, NPN 79. He died in uremia three weeks later.

A woman, aged 23 years, had blood pressure of 212/128 mm. Hg. In both eyes she showed an extreme swelling of the retina with many hemorrhages, complete absence of disc outline, a large white area between the discs and a stellate figure in the macula (fig. 9).

I have previously reported this case which is illustrative of the evanescent character of hemorrhages and exudates, for she remained under observation for 14 months. The margins of the disc became evident, the narrow arteries more defined, the retinal edema disappeared, and the macular star was reduced to a few perimacular white dots. The diagnosis was chronic glomerular nephritis with progressive hypertension. She died 15 months after she was first examined.

In nephritis the star stage may be over before the first examination, for the kidney and cardiac damage may be so severe that the patient soon dies in uremia.

For years, I have told postgraduate students that the superficial exudates and hemorrhages are of little prognostic value and that only the vessel alterations should be considered as true indices.

A 47-year-old man noticed that he could not read well with his left eye. Examination showed a low-grade edema of the retina, several spastic contractions in the narrowed arteries, a few linear hemorrhages, soft exudates to the temporal side of the disc, and an almost complete stellate figure in the macular region. The rays were short, thick, and almost white.

A month later, there was no star but innumerable, fine exudates with an increase in localized retinal edema.

When last seen, at the end of six weeks and two weeks before death, the exudate spots were fewer, with a fresh cotton-wool close to the superior temporal vein. Similar changes were present in the right eye.

At times the retinal edema will almost entirely disappear with the exception of a few diverging lines of exudate extending from the macula. This was seen in a 39-year-old man who died three months later.

An eight-year-old girl who had an astrocystoma of the vermis of the cerebellum associated with a large cyst had a pale, bilateral papilledema, a few hemorrhages but no exudates, except in a ring about the macula where they were soft gray with only a slight tendency to form a fan.

Three weeks after operation, the disc was white and less elevated. The spots in the macula were those usually found when a star is disappearing.

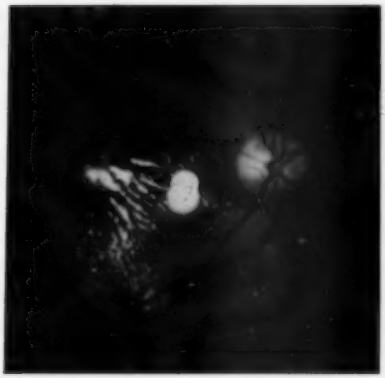

Fig. 10 (Bedell). Unusually thick macular radiations in hypertension.

A woman, aged 74 years, with a systolic blood pressure of 170 mm. Hg had vision in the right eye of 7/200. A deep, white plaque covered the macula and over it were superficial, slightly eccentric, obliquely placed exudates terminating about midway between the disc and macula (fig. 10).

Nine weeks later, the white plaques were much less conspicuous but the individual streaks were much longer and there were several fine exudates below and to the nasal side of the disc.

There was a gradual resolution with restoration of vision to 20/30.

Two and a half years after onset the

Fig. 11 (Bedell). Same patient as shown in Figure 10, two and one-half years later. No star is visible.

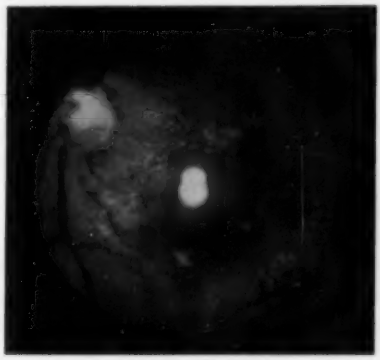

Fig. 12 (Bedell). Thrombosis of the inferior temporal vein, narrow rays.

systolic pressure was 180 mm. Hg and the fundus was negative (fig. 11).

Occasionally, exudate forms in long, thick, closely approximated, soft, yellowish, prominent bands in the lower two fifths of the region between the macula and the disc.

This was well shown in the left eye of a 69-year-old woman who died six months later of chronic glomerular nephritis with cardiac failure and hypertension.

In the right eye there was a faint suggestion of a stellate figure with much soft exudate and very evident artery and vein changes.

One of the most instructive series of the metamorphosis following a thrombosis of a retinal vein was that made of the left eye of a 62-year-old woman (fig. 12).

She was first examined four months after she noticed a change in the sight of her left eye. Her systolic blood pressure was 144 mm. Hg.

In the 13 months of observation, the blood pressure was elevated to 180 mm. Hg. The retinal hemorrhage disappeared and a primary, small star of dots was entirely dissipated. During the time of study, several unusually broad radiations developed; the largest were above the macula.

Conclusions

A stellate figure in the macular region is found in many conditions. It is often ephem-

eral and usually preceded and succeeded by other distinctive fundus signs.

The wider, the thicker, and the longer the individual rays, the more serious the prognosis as to life. When part of extensive fundus changes (edema, exudates, hemorrhages, and vascular degenerations), the star is ordinarily corroborative evidence of approaching death.

A complete star usually indicates a serious condition, although even at this late stage appropriate treatment may occasionally lead to the prolongation of life.

We can rightly conclude that the intensity of the retinal edema plus the phase of the star and the duration of the underlying causes are the important factors. When one eye has a star and the other has not, it is evident that undiscovered causes are responsible for the difference in the two eyes. All exudate lines become fine dots before complete absorption.

A better understanding of the tissue pathology of the various phases will be of great diagnostic and prognostic value. Whether the star is partial or complete, the side toward the disc is practically always the most prominent and the disc-side fan is of no differentiating value.

Stars are seen following a branch thrombosis of a retinal vein, but I have not seen them in total vein occlusions. If they are ever present in the latter condition they must be very rare.

White spots may surround a cystic degeneration of the retina, but these are not stellate figures.

In some patients the combined signs of macular star, vessel caliber changes, and hemorrhages suggest hypertension, in others diabetes, while in others they suggest papilledema, neuritis, angiomatosis retinae, or chronic nephritis. The final interpretation of each case should only be made after careful investigations, for often the fundus alterations are caused by more than one activating factor, such as a combination of hypertension and diabetes or of arteriosclerosis and diabetes.

The spokes in the wheel of exudate extending from the macular hub may be few or many, thin lines or broad elevated exudates. They are not stationary, but in a constant state of change. Accepted as danger signs, macular stars are of value, but as definitive, distinctive, particular criteria, they are fallacious.

344 State Street (6).

Ophthalmic Miniature

> Oftentimes a number of spectacles are laid before the purchasers for the purpose of their selecting what may suit them, and who generally choose glasses much too powerful, elated at the idea of seeing objects very large. . . . Those spectacles should be preferred which show objects nearest their natural state, neither enlarged nor diminished, and at the same distance, and with the same ease as could be seen before the eyes were impaired.
>
> A. Alexander: "A treatise on the nature of vision," 1833.

TREATMENT OF CYSTS OF THE IRIS WITH DIATHERMY COAGULATION*

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The literature on iris cysts is voluminous. Villard and Dejean in 1933 collected more than 230 references on this subject from the literature and added a most valuable discussion, which is still pertinent, summarizing the information of 20 years ago. Since then, although there have been many articles published, little has been added to our knowledge of this subject.

Duke-Elder's *Textbook of Ophthalmology*, although pointing out the difficulties, classifies iris cysts on an etiologic basis as follows:

1. Congenital cysts
2. Developmental cysts
   a. In the stroma
   b. In the epithelium
3. Traumatic implantation cysts
4. Retention of exudative cysts
   a. In the stroma
   b. In the epithelium
5. Degenerative cysts
6. Parasitic cysts

The recognition of iris cysts, as a rule, presents little difficulty in view of a careful history and biomicroscopic examination. The chief problem, as pointed out in a paper by Reese[10] and the discussion of it before this society in 1949, is the differentiation of certain of these cysts from malignant melanoma. That this is a real danger is shown by the fact that some of these eyes have been removed, as pointed out by Duke-Elder, Reese, and others.

It is, however, not the purpose of this paper to consider the broad field of iris cysts but rather to discuss the treatment of this condition; more specifically, to discuss treatment of the implantation or traumatic cyst.

Most authors believe that the treatment of election is the surgical removal or excision by complete iridectomy. A number of operations are described to accomplish this purpose, for example that mentioned by Duverger, Velter, and Brégeat.[4] An incision at the corneal limbus is made over the affected area and the corneal flap elevated. Under direct view the iris containing the cyst is excised. Many surgeons follow this procedure with more or less minor variations in technique.

The advantage of this operation is that it may remove the cyst completely and permits of an accurate histologic examination, which might reveal a neoplasm. The disadvantage is that it may not remove the cyst completely, especially if it is large, and recurrence may take place.

In a discussion of this subject before this society in 1937 by Ernest Krug, Verhoeff mentioned three cases in which he was fairly certain that he was not successful in removing the entire cyst, and yet there was no recurrence. He said: "In such cases it is well to attempt to remove the cysts, even if we do not succeed in excising all the epithelium. I believe that the eye can harbor a certain amount of it—possibly as a result of the operation the epithelium is incarcerated in scar tissue so that it does not proliferate."

But as Duke-Elder remarks: "Unfortunately this result is not invariable."

Another and unimportant argument against the surgical excision of an iris cyst is the mutilation of the pupil that invariably results.

Radiation by X-rays, radium, and mesothorium has been tried by some with un-

* From the Department of Ophthalmology, Northwestern University Medical School, Chicago, Illinois. Presented at the 89th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1953.

certain success. Radiation has been used after incomplete surgical removal, as discussed by Duke-Elder, and often may be the only method of treatment available when the cyst cannot be removed surgically; for example, in downgrowth of epithelium after cataract extraction. It is, however, most uncertain in its results. Recently Shaffer attempted to destroy an implantation cyst of the iris by the injection into the cyst of astatine, a substance which emanates alpha particles. The attempt was unsuccessful.

Other methods have also been tried. In 1893, Eversbusch sought to rupture the cyst by extreme contraction and dilatation of the pupil. Reid and others sought a cure by puncturing the cyst and evacuating its contents. Only occasionally has this succeeded.

The aspiration of the cyst, followed by immediate injection of a sclerosing solution such as one-percent iodine (Schöler, Alger), pure carbolic acid (Wright), or 10-percent trichloracetic acid (Lindner) has been successful but is obviously fraught with danger. Jameson in discussing Krug's paper suggested the use of two needles, one for aspiration and the other for the injection and subsequent aspiration of the sclerosing solution, as advocated by Wright.

Thilliez[18] in 1908 used electrolysis to destroy a large cyst filling two thirds of the anterior chamber. A fine needle carrying a positive charge of two ma. was inserted in the cyst for two minutes. The cyst disappeared, but recurred in six months. Another electrolytic treatment was then given with complete success. In 1914 Chaillous[2] succeeded in destroying with three electrolytic applications a large anterior chamber cyst that had recurred after incomplete surgical removal.

Safar[12] in 1935 reported the successful destruction of an iris cyst by the use of diathermic coagulation. He made a short incision of the cornea with the keratome, puncturing the cyst wall. A small Larson ball electrode with an insulated shaft was then introduced into the cyst through the wound and a diathermic current of about 50 ma. passed for a few seconds. He pointed out that it was most important not to coagulate the corneal margin of the wound, otherwise the eye is gravely damaged.

In Krug's case the patient was free from any evidence of recurrence after electric coagulation for 18 months, then the cyst recurred. A second electrocoagulation was performed. After two months the cyst returned. Glaucoma rapidly ensued and the eye was removed. Krug introduced a single needle electrode into the cyst and used a 35-ma. current for 10 seconds.

Knapp, in discussing Krug's paper, mentioned the successful destruction of an iris cyst by electrocoagulation, but unfortunately glaucoma recurred and the eye was lost, after an unsuccessful cyclodialysis. He said, "I do not feel that electrocoagulation is of great help in these cases, nor do I believe that a cyst like this could be removed by an operative procedure."

E. J. Zeiss[20] in 1949 reported the successful use of diathermy coagulation in two cases of traumatic implantation cysts of the iris. There has been no recurrence in five years in either case. Zeiss used six Kalt needles in one case and five in the other, passing them through the cornea and into the cyst. He "activated" each needle with a mild coagulation current to the point of producing a coagulation sleeve around each needle in the cornea. The postoperative reaction was moderately severe in each case, and the cornea was opaque in the area of operation. He mentioned the danger of producing cataractous lens changes and damage to the iris by this method, and his second case showed a lens opacity in the area of surgery.

Dr. T. O. Coston reported in a letter, May 13, 1953:

> In March of 1950 I coagulated an anterior chamber cyst. This was a 37-year-old man, who had a history of having suffered a perforation of the cornea with a pocket knife in 1920. Two years before I saw him he had an attack of iritis in the eye and his physician noted the presence of the cyst at that time.
>
> Six desiccating needles (as used in removing

cilia or hair roots) were passed into the anterior chamber through the cornea near the limbus margin. They were inserted at the following points: 1:00 o'clock, 3:00 o'clock, 5:00 o'clock, 6:00 o'clock, 7:00 o'clock, 8:00 o'clock. Two Kalt needles, very fine, were likewise inserted into the anterior chamber at 2:00 o'clock and at 3:30 o'clock. These needles were inserted into the anterior chamber in such a manner as to have their points in the walls of the cyst. The points, so placed, could be seen upon slightest movement of the needles. Two of the points were actually passed through the walls of the cyst and placed in such a manner as to come in contact with the posterior wall of the cyst. The needle which was inserted at 8 o'clock was passed through a small portion of the iris into the wall of the cystic area. To each of these needles a coagulation current No. 1 on the Majestic apparatus was applied. Upon application of the current to each of the needle points, there developed a grayish opacification along the course of the needle through the cornea, but a similar blanching or density of the cystic area could not be detected at the point of the needles. The coagulation current was more than once applied to each of the needles. It was noted during this procedure that the cystic areas began to decrease in size. It is possible that a part of this was a result of the puncture through the cystic wall with two of the needles. It was felt at this time that adequate coagulation and current had been applied and the needles were thus withdrawn from the anterior chamber. There was no operative complication except a minimum amount of hemorrhage from the iris at 8 o'clock. Air was injected into the anterior chamber through the Kalt needle point of insertion at 2 o'clock. The air injected gave a satisfactory depth to the anterior chamber. Atropine one-percent ointment and sodium sulfacetimide 10 percent ointment were instilled into the eye. Eye pad applied.

I attempted to insulate the shaft of the needles with a type of insulating cement which is used in re-winding generators. I did not feel that this insulation was very effective since there was some clouding of the tissue adjacent to the shaft of the needles at the time of applying the current. This was not serious and left no undue scarring afterward.

The visual acuity in this patient improved from a preoperative acuity of 20/200 to 20/50—1. A traumatic cataract, which had been present before operation, did progress over a period of a year or two to complete maturity. The cyst has shown no evidence of recurrence and there has been no other complication in the eye. I am certain that the tips of the needles did not touch the lens during the operation, but I wonder if the heat generated caused the lens opacity to progress.

Case reports

Following are three cases in which diathermic coagulation was used.

Case 1

A male student, aged 25 years, was first seen by me on September 12, 1951. Five years before a dynamite cap had exploded, producing a perforating wound of the left cornea with iris prolapse. The iris prolapse was excised and a conjunctival flap brought over the wound. The vision remained good and the eye comfortable until three weeks prior to my examination, when the eye became inflamed and sensitive to light. Examination revealed a large iris implantation cyst, in the 5-o'clock position and about five mm. in diameter. The wall was sharply defined, arising almost vertically at the pupil margin with its surface in contact with the posterior surface of the corneal vessels, one of which appeared to enter the cyst. There was some iris pigment on the anterior capsule of the lens. The vision with correction was 20/20+, the ocular tension normal, and the fundus normal.

On September 17, 1951, under local anesthesia four punctures with a very fine diathermy needle were made through the cornea into the cyst. It was most interesting to see the cyst shrivel up under the impact of 35 ma. of current for less than a second each time. Recovery was uneventful and the cyst appeared to have completely disappeared. The cornea at the site of the diathermy punctures was opaque and bloodstained.

Six weeks later the blood staining of the cornea had about disappeared and all that could be seen of the cyst was a gray flat area in which ran a few blood vessels, presumably from the iris. The vision was normal and the ocular tension normal. The lens was clear.

The patient was seen at irregular intervals and the eye showed no change until March 27, 1952, approximately seven months after the operation. At this time the cyst had recurred in a trilobular form with symptoms of ocular irritation of about one week's duration.

The cyst looked like a three-leaved clover arising from the angle at the 4-o'clock position. Several small blood-vessel loops could be seen coursing over the surface. The anterior walls were transparent, and the posterior cyst wall rested on the anterior lens capsule. The fluid within the cyst wall was somewhat yellowish and contained fine golden-brown pigment flakes moving freely in a thermal convection current similar to that of cells in the aqueous. All three lobules of the cyst connected with each other, and the cyst extended from the 2- to 5-o'clock positions, filling the anterior chamber in this area. The cyst continued to grow rapidly.

On April 23, 1952, diathermy coagulation was repeated, using three punctures carrying 35 ma. for one second each. The cyst shriveled under the current as before. The healing progressed satisfactorily. There has been no recurrence of the cyst since the last operation. The vision remained normal and no evidence of glaucoma has shown itself to date.

Case 2

A boy, aged 14 years, was first seen on March 7, 1952. At the age of four years, his right eye had

been injured with a knife. One year later several cilia in the anterior chamber were removed. At the age of 10 years, a small cyst of the iris was diagnosed and medical treatment given which improved the eye. The nature of this treatment is unknown. One month before admission the eye became sensitive to light.

At the time of examination the right eye was inflamed and sensitive to light. The ocular examination showed a large implantation cyst filling the temple side of the anterior chamber from the 7- to 11-o'clock positions extending to the pupil margin. The pupil was distorted toward the temple side and a secondary cataractous membrane was present.

The anterior surface of the cyst was in close contact with the posterior surface of the cornea, producing localized epithelial edema. The cyst contained clear fluid and the base consisted of fibrous tissue interspersed with uveal pigment. The red reflex was only fair due to the cataractous membrane, and the vision was reduced to hand movements. The ocular tension was normal.

On March 13, 1952, under local anesthesia the cyst was destroyed with a fine diathermy needle, coagulation at 35 ma. for two seconds. There was little postoperative reaction and the ocular tension remained normal. The corneal wound showed blood-staining and white coagulation which became scarred over an area of about 2 mm. There has been no recurrence to date.

CASE 3

A physicist, aged 33 years, had a perforating injury of his left eye on August 8, 1952, as the result of an explosion in his laboratory. A fragment of glass caused a V-shaped laceration at the limbus of the left cornea at the 3-o'clock position. His ophthalmologist skillfully sutured the cornea and covered the area with a Van Lint flap. On September 10th it was noticed that the scar was becoming fistulous.

My examination of October 27th disclosed a moderately congested left eye with a small fistula in the cornea at the 3-o'clock position, covered with a thin epithelial and vesicular scar. The anterior chamber was normal in depth, the root of the iris was incarcerated in the fistula. The lens was clear, and the corrected vision 20/20. The ocular tension was: R.E., 20; L.E., 23 mm. Hg (Schiøtz).

On November 11th a small keratome incision was made, the synechia freed, and the vesicular surface of the fistula cauterized with diathermy. The convalescence was somewhat stormy and the tension became elevated (55 mm. Hg, Schiøtz). This responded to miotics and the tension remained normal until December 2nd, when an acute attack of glaucoma occurred; this was controlled with DFP for three weeks.

At this time a small iris cyst was discovered at the 3-o'clock position and further vesiculation of the scar had developed. The tension rose to 32 mm. Hg in the course of the next few days and it was decided to coagulate the cyst with diathermy and perform a cyclodialysis.

On December 31, 1952, a fine electrolysis needle was inserted into the cyst and touched with the diathermy electrode carrying a current of 10 ma. for one second. The cyst collapsed. A cyclodialysis in the 1-o'clock position was then performed. In the next few days the tension rose rapidly to 65 mm. Hg and the characteristic picture of acute glaucoma ensued. After a difficult time with miotic and local cortisone treatment the tension returned to normal and the congestion subsided.

On January 15, 1953, the cyclodialysis was patent as seen with the gonioscope. For the next two weeks the tension tended to become elevated but this was controlled with eserine when needed. The scar of the cornea was firm and no longer vesicular. The iris at the site of the cyst was atrophic with fine scattered pigmentation. The pupil was drawn toward the 4-o'clock position. The vision with correction was 20/20 and the patient was comfortable. Miotics were discontinued and there was no further rise in tension.

On February 2, 1953, the tension measured 20 mm. Hg (Schiøtz) and did not rise after mydriasis with euphthalmine. A recent report from his physician states that the patient is entirely comfortable and has normal vision and tension.

COMMENT

Traumatic implantation cysts are not rare and, if allowed to remain, they continue to develop and eventually destroy vision, chiefly because of secondary glaucoma. Efforts are therefore made to remove or destroy them, often unsuccessfully. The methods that have been used are (1) surgery, (2) radiation, (3) chemicals, (4) electrocoagulation, and (5) electrolysis. There are case reports in the literature which cite successes and failures with each of these methods, and the problem therefore appears to be not yet completely solved.

The surgical removal of iris cysts can be quite difficult and hazardous, especially in aphakic cases, and they tend to recur. Radiation with radium or X rays is uncertain in its results and difficult to pinpoint and to control, so that damage to other parts of the eye, especially the lens if present, is a genuine hazard. The injection of sclerosing chemicals into the cyst is difficult, and if the solution gets through the cyst wall, it can do considerable damage. Both Arnold Knapp and John Wheeler in discussing Alger's paper described cases in which iodine penetrated into the aqueous chamber, resulting

in an alarming, though temporary, opacification of the cornea. Knapp said:

"My associates and I passed some anxious weeks watching to see whether the corneal cloudiness would clear up and whether the lens would suffer. Fortunately, the corneal opacity did clear up, and the lens was not injured, but that danger has to be remembered in the injection of iodine into the anterior chamber."

Electrocoagulation therefore remains as a method that offers an opportunity to destroy the cyst with less chance of injuring the rest of the eye. As a survey of the literature and reports of personal cases show, there have been a few failures, but also promising successes. At the moment, therefore, diathermy coagulation seems to be the best method at hand. Electrolysis, which has a similar action, can be included. It may be that electrolysis, after further trial, will prove in the end to be superior to diathermy coagulation.

It is, of course, obvious that the lens, if present, can be damaged by contact with the diathermy needle. However, if this danger is borne in mind, experience has shown that it can be avoided.

There is a variation in technique as reported by the various authors, and this point will require further study. The question of whether it is better to insert several small needles at once into the cyst and then touch each one with the coagulating current, or to insert repeatedly into the cyst, through the cornea, a fine needle carrying the current, needs experimentation.

The thought at once occurs, should not these needles be insulated to avoid corneal injury? Attempts have been made to do this by applying an insulating lacquer to the needles, but not enough experience is available to say whether or not it is worth while.

It can be argued, and properly so, that an incision in the corneal limbus, elevation of the corneal flap, and destroying the cyst with diathermy coagulation, under direct view, as described by Safar,[12] offers the cleanest way to avoid damage to the cornea. However, this is hazardous to do in an aphakic eye. In my three cases the damage and subsequent opacification of the cornea was trivial, and since the scar was at the limbus, was unimportant to vision.

The technique is simple to use and can be repeated if necessary. Secondary glaucoma appears to be the chief postoperative complication, and may require surgery to correct. Its mechanism may be on a neurovascular basis as the result of irritation to the iris, assisted by local injury to the filtration angle at the site of the punctures.

Summary and Conclusions

1. Three cases of traumatic implantation cyst of the iris successfully destroyed with diathermy coagulation are described.

2. Other methods, which have been used with more or less success, are discussed.

3. It is considered that diathermy coagulation, and probably electrolysis, offers the simplest and most efficient method known at the moment for destroying iris cysts.

4. Experimental work should be done and more experience obtained in order to develop the proper technique and dosages required.

5. The method described would seem to be applicable to other forms (except downgrowth of epithelium lining the anterior chamber) of iris cysts.

700 North Michigan Avenue (11).

References

1. Alger, E. M.: Large implantation cyst of the iris, treated by aspiration and injection of iodine. Arch. Ophth., **7**:984, 1932.

2. Chaillous, M. J.: Treatment of transparent cysts of the anterior chamber by electrolysis. Soc. française d'opht., **31**:450, 1914.

3. Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1941, v. 3, pp. 2432-45.

4. Duverger, C., Velter, E., and Brégeat, P.: Therapeutique chirurgicale ophtalmologie. Paris. Masson et Cie, 1950, p. 236.

5. Eversbusch: Cited by Duke-Elder.[3]

6. Jameson, P. C.: Discussion of Krug,[4] p. 420.
7. Knapp, A.: Discussion of Krug,[4] p. 420; Discussion of Alger,[1] p. 985.
8. Krug, E. F.: Cyst of the posterior chamber. Am. J. Ophth., **21**:412, 1938.
9. Lindner, K. D.: In Meller's Ophthalmic Surgery. 6th Edition by J. Böck. New York, Blakiston, 1953, p. 389.
10. Reese, A. B.: Spontaneous cysts of the ciliary body simulating neoplasms. Tr. Am. Ophth. Soc., **47**: 138, 1949.
11. Reid, C.: Cited by Duke-Elder.[5]
12. Safar, K.: In Meller's Ophthalmic Surgery. 6th Edition by J. Böck. New York, Blakiston, 1953, p. 390.
13. Schöler, F.: Cited by Duke-Elder.[5]
14. Shaffer, R. N.: Alpha irradiation: Effect of astatine on the anterior segment and on an epithelial cyst. Candidate's thesis. Tr. Am. Ophth. Soc., **50**:607, 1952.
15. Thilliez: Treatment of iris cysts by electrolysis. Soc. française d'opht., *25*:490, 1908.
16. Verhoeff, F. H.: Discussion of Krug,[4] p. 419.
17. Villard, H., and C. Dejean: Cysts of the iris. Arch. d'ophtal., **50**:91, 194, *272*, 1933.
18. Wheeler, J.: Discussion of Alger,[1] p. 985.
19. Wright, R. E.: Cited by Duke-Elder.[5]
20. Zeiss, E. J.: Posttraumatic anterior chamber cysts. Am. J. Ophth., **34**:151, 1951.

BARON WENZEL'S OPERATION*

For the extraction of cataract: With an exhibition of such instruments as were used

Burton Chance, M.D.
Philadelphia, Pennsylvania

The reviving of the use of scissors with which to lengthen the line of incision at the sclerocorneal junction for the extraction of the lens in cataract operation has reopened in the history of ophthalmic surgery the chapter long closed by the facile method inaugurated by von Graefe.

It has been said that Daviel opened the cornea in his first case with scissors. Later on he employed a modified scalpel and then lengthened the incision with specially devised scissors, one pair bent to the right, the other to the left, to enlarge the opening in order to facilitate the extrusion of the lens.

In the hope of finding a Daviel scissors, a search was made through the collection of instruments at the College of Physicians of Philadelphia, but none resembling them was found. In a neat, much worn, leather-covered case was found a pair which were much stouter than any depicted as of Daviel's scissors. The search was discontinued because in the case containing the scissors was a collection of Baron Wenzel's cataract instruments, and it is of these and their use that this brief communication is to be made. The Baron described them in his *memoire*† published in 1786 in French; this was lucidly translated by Ware in 1791.

The main instrument is a keratome in the form of an isosceles triangle, the blade of which is about 1.5 inches long and three-eighths of an inch in width at its base. The second instrument is double pointed and ivory handled—a rod of about two inches' length, bent at right angles at the tip end with, at the other end, a "Daviel" scoop or curette, four or five short wire rods, and a pair of small forceps. In Wenzel's *memoire*

* Presented at the 89th annual meeting of the American Ophthalmological Society, Hot Springs, Virginia, May, 1953.

† *Traité de la cataracte, avec des observations qui prouvent la nécessité d'inciser la cornée transparente et la capsule du crystallin, d'une manière diverse, selon les différentes expèces de cataractes*, Paris, 1786.

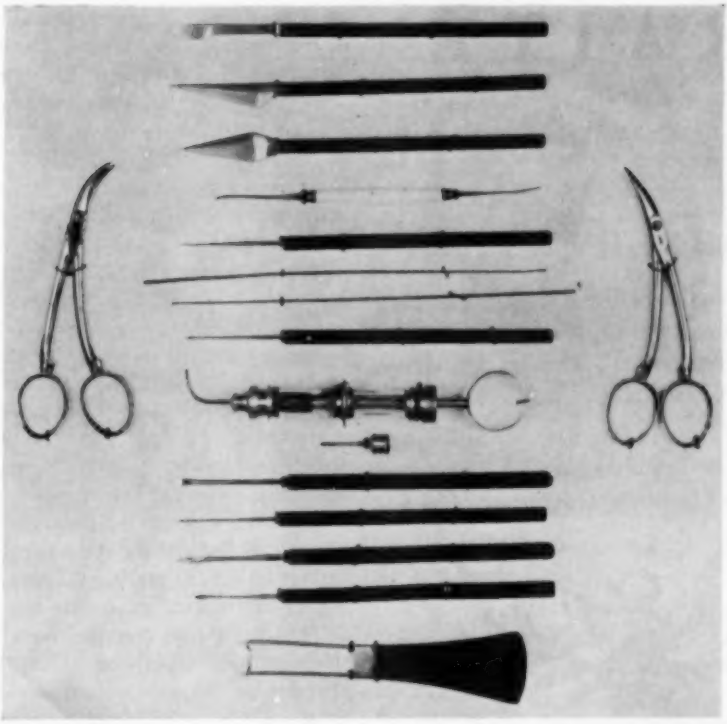


Fig. 1 (Chance). Instruments from the museum collection, College of Physicians of Philadelphia, believed to have been from the armamentarium of Professor Physick.

the procedure of their use is minutely described. Figures 3 and 4 are from the *memoire*.

Wenzel did not use a fixation forceps nor speculum, but trusted in steadying the eye with his fingers. No anesthetic was used; the surgeon sought to quiet the eye at the time of operation simply by his own manipulations. The operation was conducted with the patient sitting in a particular chair; an assistant standing behind rested the patient's head against his chest and carefully raised the upper lid, pressing gently on the globe, making it possible for the surgeon to further steady the eye with his own fingers. The surgeon sat in another chair somewhat higher than that of the patient, his foot on a stool so that his knee was bent, with the keratome in his right hand when operating on the patient's left eye, and with his bent elbow resting on his knee. The right eye was operated on with his left hand.

The keratome was "plunged," at the upper sclerocorneal junction, into the aqueous chamber and straightway carried therein. At the pupil the keratome was inclined slightly so that the capsule of the crystalline might be lacerated; the keratome was then withdrawn from the pupil and pushed to the lower angle of the chamber a short distance from the junction, and a crescentic incision made into the cornea.

In the passage of the knife through the chamber, care was taken lest the sheet of the iris fall across the blade, which sometimes seriously lacerated it. In other cases the surgeon, by massaging the cornea with the closed lid, could replace the membrane

TRAITÉ
DE
LA CATARACTE,
AVEC
DES OBSERVATIONS

Qui prouvent la néceſſité d'inciſer la cornée tranſparente & la capſule du cryſtallin, d'une manière diverſe, ſelon les différentes eſpèces de Cataractes:

Par M. De Wenzel, fils, Baron du S.-Empire, Médecin de la Faculté de Nancy, & Docteur-Régent de la Faculté de Médecine en l'Univerſité de Paris.

A PARIS,

Chez P. J. Duplain, Libraire, cour du Commerce, rue de l'ancienne Comédie Françoiſe.

M. DCC. LXXXVI

Avec Approbation, & Privilège du Roi.

Fig. 2 (Chance). Facsimile of Wenzel's title page.

and the operation was continued successfully.

To avoid such accident, the keratome with a blunt edge was used. Carefully withdrawing the keratome, the surgeon began by pressure on both the upper and lower aspects of the globe; the crystalline was dislocated and extruded through the pupillary space. When the iris was found to be adherent to the crystalline structure, Wenzel employed a gold rod similar to those in the set shown.

In some cases the bent, pointed rod, the forerunner of the later sharp-pointed cystotome, was used; in others, it was necessary to employ the scoop-curette. The border of the crystalline fitted into the bowl of the scoop. The scoop also was used to gather any crystalline fragments found in the aqueous. At times, as might be expected by us today, there was a prolapse of the iris which the author denominated as "staphyloma."

The small forceps depicted might be used to further loosen the pupil and to seize the capsule and completely withdraw the crystalline—a true extraction of the crystalline.

We can imagine the difficulty and the seriousness in the case of a contracted pupil. It was not until about 1800 that attempts were made to widen the pupil as Himly employed solutions of hyoscyamus, and from that time on mydriasis was practiced.

When all was quieted the patient was led back to his bed and the eye was covered with a sheet of lint; after 10 or 12 days, full healing of the surgical procedure was expected.

Thus the Baron described very briefly an uncomplicated operation. The remaining chapters of his *memoire* are devoted to the details attending more serious incidents and complications well worthy of close reading by experienced modern surgeons.

The set of instruments shown was long forgotten. It is not known to whom in America this case originally belonged. It was among the treasures collected by the late Dr. Fenton and contributed to me. It was donated to the Museum at the College of Physicians, and remained obscure until the search for the Daviel scissors. It probably was Physick's own, as Wenzel's operation was that which he pursued for many years. Physick, as well as the Baron, dreaded the consequences of couching and reclination.

In a medical student's notebook* of Physick's lectures on surgery at the University of Pennsylvania the instruments and the

* "Notes on Surgery Taken from the Lectures of Philip Syng Physick, M.D., Professor of Surgery in the University of Pennsylvania, by William Elmer, Student in Medicine, during the Winters 1808-9-10."

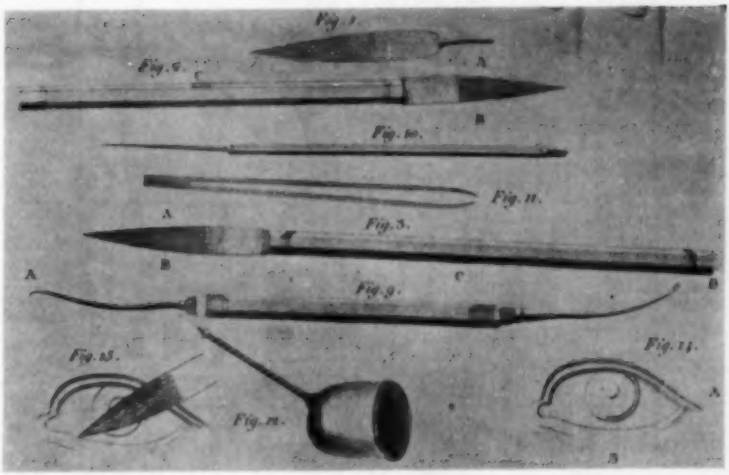


Fig. 3 Chance). Wenzel's cataract instruments. (Reproduced from Wenzel's *Traité de la cataracte.*)

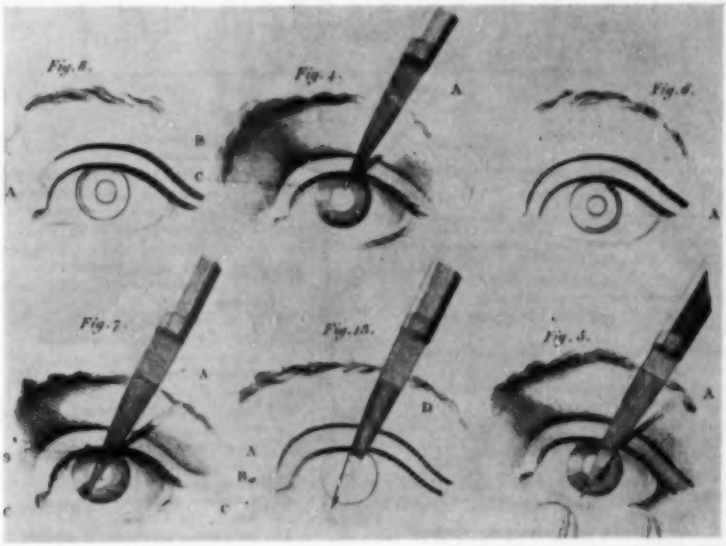
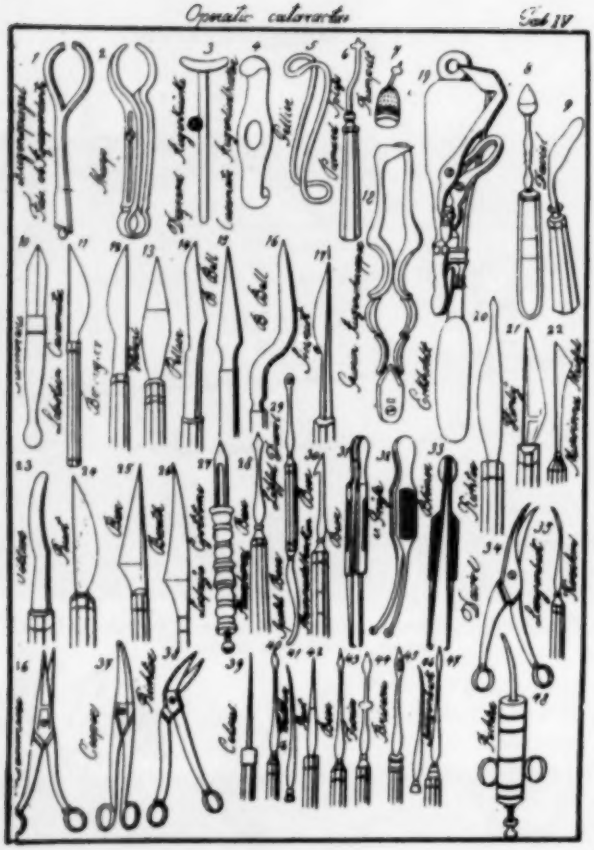


Fig. 4 (Chance). The Wenzel operation. (Reproduced from Wenzel's *Traité de la cataracte.*)



Tab. IV.

Operatio cataractae.

Fig. 1. Fabricius ab Aquapendente, Augenspiegel zum Offenhalten der Augen.
- 2. Sharp, Augenspiegel.
- 3. Degrand, Augenkrücke.
- 4. Cusomata, Augenliedhalter von Silberblech, um das untere Augenlied herabzuziehen.
- 5. Pellier de Quincy, Augenliedhalter von Silberdraht.
- 6. Pamard, Spiess zur Fixirung des Augapfels.
- 7. Rampelli, silberner Fingerhut mit Stahlspiess zu demselben Zwecke.
  - * Ollenroth, Fingerring zu demselben Zwecke. (s. Suppl. Taf. Fig. 8.)
- 8. 9. Daviel, Werkzeug zur Eröffnung der Hornhaut.
- 10. Santorelli, Staarmesser.
- 11. Lobstein-Cusomata, Staarmesser.
- 12. Berenger, Staarmesser.
- 13. Wenzel, Staarmesser.
- 14. Pellier, Staarmesser.
- 15. 16. B. Bell, Starrmesser.
- 17. Siegrist, Staarmesser.
- 18. Guerin, Augenschnepper zur Eröffnung der Hornhaut.
- 19. Eckholdt, Augenschnepper zu demselben Zwecke.
- 20. Richter, Staarmesser.
- 21. Himly, Staarmesser.
- 22. Marsiana, Meissel zur Eröffnung der Linsenkapsel.
- 23. Pellier, Staarmesser.
- 24. Bust, Staarmesser.
- Fig. 25. Beer, Staarmesser.
- 26. Barth, Staarmesser.
  - * Marsiana, Staarmesser. (s. Suppl. T. Fig. 4.)
  - ** Pope, Staarmesser. (s. Suppl. T. Fig. 3.)
  - *** Arnemann, Staarmesser. (s. Suppl. T. Fig. 5.)
  - **** Popé, Staarmesser und Nadel. (s. Suppl. T. Fig. 7.)
- 27. Lafaye, federndes Cystitome z. Eröffnung d. Linsenkapsel.
- 28. Beer, Staarlanze, zu demselben Zweck.
- 29. Beer, Spatel und Daviel, Löffel von Silber zur Entfernung der Linse.
- 30. Beer, Staarnadelhaken eben dazu.
- 31. Beer, Hakenpincette zu Operationen im Augapfel.
- 32. v. Gräfe, Augenpincette.
- 33. Blomer, Hakenpincette.
- 34. Daviel, Augenscheere.
- 35. Langenbeck, Keratom zur Discision der Linse.
- 36. Santorelli, Augenscheere.
- 37. Cooper, Augenscheere.
- 38. Richter, Kniescheere.
- 39. Celsus, Staarnadel von Silber.
- 40. v. Walther, gekrümmte Staarnadel.
- 41. Dieselbe von der Seite.
- 42. Bust, gerade Staarnadel, in der Mitte blau angelaufen.
- 43. Bier, Staarnadel.
- 44. Ferrio, Staarnadel.
- 45. Brisseau, gerade Staarnadel mit ausgehohlter Spitze.
- 46. 47. Langenbeck, gekrümmte Staarnadel.
- 48. Richter, Augenspritze.
- ***** Schmidt, Staarnadel. (s. Supp. T. Fig. 6.)

Fig. 5 (Chance). From Fritze's *Miniature Armamentarium*, Berlin, 1836.

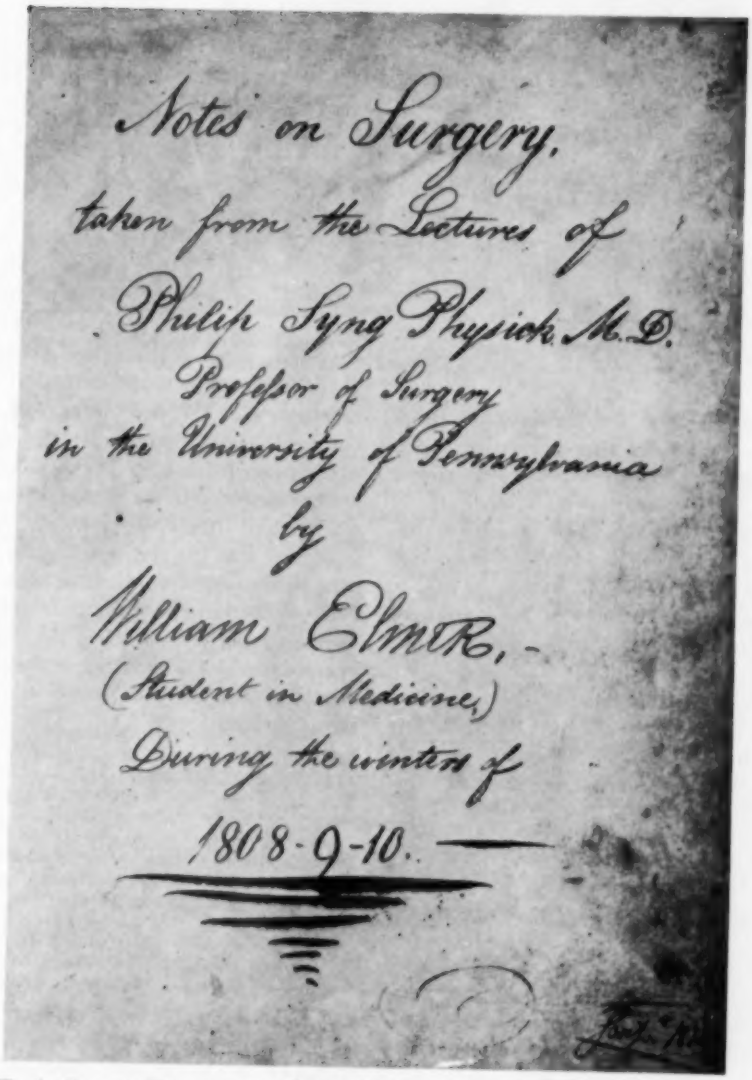


Fig. 6 (Chance). Title page of medical student's notes of Physick's lectures in surgery. (Reproduced by the kindness of Walter G. Elmer, M.D.)

Fig. 7 (Chance). Student's sketches of Physick's description of Wenzel's instruments. (Reproduced by the kindness of Walter G. Elmer, M.D.)

details of operation are described exactly as though copied from the original *memoire*. The page is accompanied by pen-and-ink sketches of the instruments. According to these notes, Physick's case contained scissors. The small syringe in the collection suggested to me that it might have been used to flush the aqueous chamber to free it from the fragments of lens remaining at the extraction.

Physick's skill must have been truly great. His last operation, performed shortly before his death in 1837, was successful. The student's notes emphasized the delicacy of Wenzel's operation. As late as 1837, Littell, in his small book, feared that the operation would never become successful and his opinion was repeated in the second edition of his work published in 1846.

Wenzel was the son of a famous wandering cataract operator who traveled all over Europe, operating in town after town, never attending the patients after the day of the operation. The son, the author of the *memoire*, was well established in Paris. He never used scissors.

317 South 15th Street (2).

Ophthalmic Miniature

Should one eye only be affected, the operation for the cataract should be declined, as one eye may be fully sufficient for tolerable vision, and by the operation on one eye, the focus of vision would become dissimilar in the eyes, and the patient derive very little benefit from the operation.

J. B. Serny, M.D., Oculist (1809),

"A treatise on local inflammation, more particularly applied to diseases of the eye."

ALBRECHT VON GRAEFE: THE MAN IN HIS TIME

EGON V. ULLMAN, M.D.
Portland, Oregon

PREFACE

"I attach to common everyday facts, either hidden or plain to be seen," Balzac once said, "and to the acts of individual lives, and to their causes and principles the importance which most historians . . . ascribe only to great events of public national life!" To him the "dry and discouraging episodes called History" did not amount to much without a history of "private *maintien*."

Albrecht von Graefe's life has been written many times, but in the manner that biographies of royal personages used to be written long ago when kings and queens were either stainless ghosts or resonant statues, "private *maintien*" left out or barely hinted at, achievements looming large.

As Albrecht von Graefe was the greatest ophthalmologist of the 19th century, his achievements loomed large enough. But they have been dealt with by many a fond and faithful scholar, and I do not think I disoblige his memory by trying to make a portrait of the human being who did great deeds rather than attempting to draw a landscape of the high peaks of his work and assuming that the man, like the bear who went over the mountain, is on the other side.

AUTHOR'S NOTE

While writing this article I asked assistance and co-operation from the German Ophthalmological Society of Heidelberg. Dr. E. Engelking, Professor of Ophthalmology at the University of Heidelberg, Custodian of the Graefe Museum, replied to my request for new or unpublished material with great courtesy and generosity. Through him I was able to secure copies of photographs, personal letters, and documents, many of which are used herein. I am deeply grateful to Dr. Engelking for his help and to the Graefe Museum for permission to use this valuable accumulation.

Young Dr. Peter Reimann, of the Heidelberg Medical Policlinic, did considerable spade work for me, deciphering and extracting data from dozens of Graefe letters, and I wish to thank him.

Miss Bertha Hallam and her staff at the Oregon Medical School Library let me, without reprimand, keep Graefe literature so long it sprouted, and I want to say that was very kind.

It might be interesting to mention here that I was unable, hard as I tried, to find a living descendant of the great old von Graefe family. Dr. von Brunn, Professor of History of Medicine of the Karl Sudhoff Institute in Leipzig, wrote me last year, "I knew the son of Albrecht von Graefe, who was a gentleman farmer in Mecklenberg. . . . He had a son who if living would be about 50 years of age. This man, since 1945, when the old Graefe country estate in East Prussia, *Goldebee*, was taken out of the hands of the family and parcelled off, has disappeared." If continents and languages go down, it is not so strange that a famous and ancient clan should exist and then not. So far as it is known, no von Graefe, male or female, is alive today.

This work was aided by a grant from the Kaiser Foundation.

I

At about the time the third son of Karl Ferdinand von Graefe, Professor of Surgery at the Berlin University, was born, a famous woman was engaged in writing a word or two of reminiscence about German doctors. This celebrated female, Madame Johanna Schopenhauer—mother to as surly a cub as ever saw the light of day—wrote that when she was a girl the physician's main characteristic was hoary age. He could no more be imagined as a tumbling boy or hot-cheeked youth or enterprising man, than could the Pontiff or St. Paul. Herr Doctor? Don't over-reach yourself. His title is "Excellency" and only his most cardinal friends or metaphysical foes dare address him otherwise.

He wears a curly white wig, so weighty that it is a good thing some of the burden rests upon his powder-whitened shoulders, or it might prove too much of a load for his head. This latter is already as heavy as a bucket of sand with omniscience. Can you not tell what a wise man he is, just by looking at him? See that bright red coat, fenced with gold lace! those ruffles! that oversize frill of shirt sticking out like a sail-fin's wing! his shining hose! knee pants! the

buckles on those small-heeled shoes! the hat, if you please! lowcrowned, cocked, too little to fit anybody but a pinhead, meant only to be carried under the arm, not worn. Most particularly, observe that cane! without a knob but with a broad comfortable hilt intended to be used as a *prop for his chin* whenever his Excellency has occasion to think! "He did not do it very often," wrote Madame Schopenhauer.

Had she lived to see Albrecht von Graefe open his clinic on the Karlstrasse, she would have seen as young a physician as heart could desire; one of the youngest and most devoted ever to start practice. Within her life span, the bedizened old bluffer smelling of the Middle Ages, with the dirt of the Renaissance under his fingernails, who had been for many a decade reshaping, was to appear transformed into the manner of doctor ourselves know today, and the conversion from old to new be accomplished. But Madame Schopenhauer died just a little too soon for that event, when Albrecht von Graefe was a boy of 10, nine years before he emerged as one of the first and fairest examples of modern physician.

His degree was almost the only thing he ever had to earn, and even that did not cost him what it costs many another. Not to Albrecht von Graefe returned in after life the nightmare of the examination he could not pass. A born scholar, he could pass them all, for learning came easy to him. What did not? The stars—perhaps because they were going to be so very hard on him at the end—were prodigiously good to Albrecht von Graefe at the beginning.

First, the day of his birth, May 28th, could not have been improved upon, for it was ideal. The year, 1828, was ideal too. Were there no wars then? you ask. Dissensions, call them, cock fights, ladies' hair-pullings, duels. The Czar of all the Russias and Sultan Mahmoud went a round or two by proxy; Bolivar and Peadella sparred a little; in France—in Africa—but it was hop-o'-my-thumb scrimmaging then, in comparison with later, and any number of people supposed that year and world the best of all possible years and worlds. In America they thought so, assuredly, with their 1828 novelties—their railroad, steamboat, fire bricks, straw paper, planing mill, lithographic press, varnish—all brand-new toys with the price tags still on. They thought so certainly in England, except perhaps Thomas Carlyle, who had a sour stomach. They thought so in France, in Paris especially, for that was the year the omnibuses began to run there.

The year after Beethoven died, Albrecht von Graefe was born at the Haunt of Finches, a luxurious summer house in the Tiergarten of Berlin, the gift of a prince to his father, Ferdinand von Graefe, teacher of note, general in the Prussian Army and court physician, to be known as the founder of modern plastic surgery. Albrecht's mother, born Augusta Alten, heiress and belle, though an old married lady of not less than 34 at the time of his birth, was still beautiful and gay. She loved Haunt of Finches and claimed to be sorry when autumn came and the season called the fashionable back to town. But fat and sassy Albrecht had got a good start, at least, she said, when sighing she gave him a squeeze and turned him over to his own nurse, a young and pretty Vierländer in a stiff cap and wide apron, who had nothing to do but take care of the baby (godfathered by a Minister of State) in the nursery of the family mansion at 48 Behrenstrasse, one of the fashionable streets in Berlin.

It was a great city, though many of its thoroughfares were not paved, and housing, drainage, and water supplies were bad. Some said the air was not brisk enough and this was thought to account for the lung troubles that abounded. Actually, as everyone knew, there were two classes of people, the weak and the strong, and if the former did not have something the matter with their chests, they had something the matter somewhere else, as God willed it.

Brisk or not, the air of Berlin greatly

agreed with the new arrival, the air of his native land, that had been good enough for Goethe for 79 years already and would be good enough another four, and Albrecht grew big and tall.

At 10 years of age, he started school at the French Gymnasium where it was almost impossible not to learn to speak, read, and write French like a native—to some its chief advantage—and where otherwise the usual German high-school curriculum was taught. There a dry young man with a green old name, Molière, instructed him in religion.

One sleety February morning when Albrecht approached the school, he was met by a schoolmate, son of an attaché at the Imperial Palace. The boy was pale and trembling. "Oh, Albrecht," he exclaimed, "last night the Mistress of Her Majesty's Bed Chamber saw the White Lady!" Albrecht looked shocked. Everyone knew that the White Lady was one of the oldest ghosts of the realm, the earthbound spirit of a certain Countess Agnes of Orlamunde who had murdered her two children in order to marry a handsome young Brandenburg elector named Albert some hundreds of years before. When she appeared, it meant that a Hohenzollern king was rapidly approaching his end.

That evening Albrecht's father laughed when he heard the rumor. He said the White Lady was a shaft of moonlight in the Palace corridor or somebody's royal underwear, and added that the King had never looked better in his life.

Albrecht felt ashamed of believing such nonsense. Five months after, however, the ruling monarch, Fredrick William the Third, of the house of Hohenzollern, did indeed die though he was still a young man in seeming good health. Then Albrecht remembered the apparition and longed to inquire just how responsible the White Lady might have been for the melancholy event. It seemed like such a coincidence! But he was shy of broaching the subject, the busy court physician was seldom at home and a month later there was no chance of talking to him on that topic or

Fig. 1 (Ullman). Albrecht von Graefe, aged 12 years. From a pencil sketch by his sister Ottilie.

any other at all, again, for Albrecht's father was dead. He died on June 7, 1840, after a brief illness, when his youngest son was 12 years old, leaving each and every member of his family well provided for, for the rest of their lives, and all sensible of irreparable loss.

Dr. Karl Ferdinand von Graefe at the age of 23 had been personal physician to Duke Alexius of Anhalt-Bernburg. Then he became the first director of the Surgical Clinic at Berlin and had held this post for 30 years at the time of his death. He wrote the *Encyclopaedic Dictionary of the Medical Sciences*, founded the first journal of surgery and ophthalmology, was a practicing ophthalmologist, and two years before Albrecht's birth performed the first cataract operation in Germany with a corneal cut directed upward. Up until 1827 he had accounted for 431 eye patients and was famous enough so that visitors, who went to the trouble of asking for diets and prescriptions at the

same time, came expressly to watch him perform eye operations. His capabilities would have been signalized and remembered for a long while if he had been followed by a nonentity. But his unhappy fate was to be succeeded by a genius, Dieffenbach, whose passion lay in the field of general surgery, so Dr. von Graefe the elder, and with him his many achievements, particularly those in ophthalmology, was soon forgotten.

Albrecht von Graefe, at 15, was the youngest boy ever to graduate from the French Gymnasium. Lacking a year of being old enough to enter the university, he put in the time studying mathematics and physics with a Professor Goepel, and dabbling with chemistry under the guidance of Professor Ramelsburg. His mother consented to have a small laboratory built and fitted up for him but she wondered if his head wasn't a little too old for his young shoulders. Ought he not to enjoy himself more? His sister Ottilie and cousin Mathilde agreed that while it was true he might not be so fond of society's amusements as other young scions they knew, and was partial to study, yet he was not a grind, they said. He liked to hike and climb mountains. He had lots of friends of his own age and station. He was high-spirited and full of foolishness. Besides, too much study would have made him pale and languid and he was the picture of health! Thus the two girls pleaded his cause and his mother, reassured, let him follow his inclinations.

Before entering the great German school he had to be confirmed in the church, and this done, he enrolled as the youngest pupil at the University of Berlin. It was, at that time, itself a young institution, but 35 years old, and quite scattered, the administrative offices in a royal palace, the library in a street some distance away, the dissecting room and medical college in still another district. Baron Humboldt's great mineral cabinet, the best in Europe, reposed at the Museum of Zoology. Many departments and schools made up the university, and over all, whether near or far, rested a certain unmistakable atmosphere, as tarnish upon silver, a cloudiness breathed by Frederich Hegel, who had died 13 years before. The philosopher, supposed to have been understood by but one of his fellow creatures "and even he has not understood me," credited with having said, "No man is a hero to his valet," and whose work he himself described as "an attempt to teach philosophy to speak in German" was, to *ancilla theologiae* in Germany, what Goethe was to literature and Beethoven to music. He was, in short, the Grand Cham, holding the chair of philosophy at the university from 1818 until he died of cholera in 1831 at the age of 61. When Albrecht von Graefe attended the university, the Hegelian influence was still thick enough to cut with a knife. Because of the rising science of psychology and due to the reaction against the speculative method, to name but two of many reasons, Hegelianism was to be short lived but our von Graefe got a dose of it full strength, to his no great harm.

He signed up for a course in anatomy under versatile Dr. Johannes Müller, who taught decriptive and comparative anatomy and embryology. The founder of scientific medicine in Germany and the country's greatest physiologist was then 43 years of age. Temperamentally a mystic, and a "vitalist" in theory, he was a big imposing man who could charm the birds out of the trees.

"Gentlemen—students—," he said one day in the midst of demonstrating the embryonic circulation which was drawn in two colors on an easel, "my grandfather, when in England some 60 years ago, made the acquaintance of one Dr. Moore. 'I am sure,' said this doctor to my grandfather, 'that God made the world, and what's more I am *positive* that he made man, too!' 'How so?' said my grandfather. 'Because,' said this Dr. Moore, 'in my profession I have had to study a good deal of anatomy, and for this reason I have come to the conclusion that, had anyone *else* made man, he would have been bigger than an observation tower, for,' he said, 'there

would have been no room to put in all the little vessels and small parts we are composed of in any giant less than 800 feet high.'"

In the year 1840 Johannes Müller announced that no one could ever expect to measure the velocity of a nervous impulse, and a decade later his pupil Helmholtz had accomplished it, so he could be, and sometimes was, mistaken, but he was a great teacher and many of his students, Rudolph Virchow among them, are said to have derived the inspiration for their life's work from him. He was called "The Olympian" by his colleagues. Albrecht von Graefe in his first two years as a medical student thought the name would have applied as well to almost any one of his teachers, for that was a Golden Age at the university.

Hegel's successor, Michelet, taught him logic and philosophy.

Johann Friedrich Dieffenbach, the surgeon who took Karl Ferdinand von Graefe's place at the Charitè, taught him surgery and ophthalmology. This man had started up several wrong paths. He operated for strabismus by cutting the tendons of the eye muscles—from turning in, the eye turned outward, or vice versa. This ridiculous operation possibly led him to attempt the gruesome cutting out of a wedge of the tongue for stammering, a procedure both useless and catastrophic, but he had many patients and in 1841 published a paper called "The cure of stammering." Still, Dieffenbach had noble qualities as a teacher. Ernst Wilhelm von Brücke, whose book on *Artistic Anatomy* (1892) is still today one of the most attractive books ever written on the subject, and Emile Du Bois Reymond, whose main work was on the reactions of tissues to electricity, taught physiology.

When Albrecht signed up for a course in internal medicine called symptomatology under Ludwig Traube, a small Jew with soft garnet-colored eyes, student of morbid anatomy and physiology and a pioneer in experimental pathology, whose great interest

Fig. 2 (Ullman). *In the Harz Mountains.* Albrecht von Graefe, aged 16 years, sketched by his sister Ottilie.

was fever and who introduced the thermometer into the clinic, he little thought that this young man would someday be his own physician. (Not only that, he would be present at Graefe's death . . . and close his eyes.) Traube was the first man in Germany to experiment with animals, cutting the vagus nerves of dogs to see what effect that would have on breathing, and wrote a paper on the "Symptoms of suffocation."

Friedrich Schlemm, who discovered and described Schlemm's canal, taught the art of dissection with a zeal that bordered on passion.

Handsome plump Moritz Heinrich Romberg, whose "positive Romberg" we still use today, lectured on internal medicine and neurology. He had been teaching six years when Albrecht took classes with him, and was famous for having made the first real advance in the interpretation of ataxia and

Fig. 3 (Ullman). *The university student.* Albrecht von Graefe, aged 17 years, sketched by his sister Ottilie.

writing the first formal treatise on nervous diseases.

Yes, Albrecht von Graefe, an idealistic boy, looking for excellence everywhere, would have pronounced them all Olympian—except two—Juengken, the bull-headed old beau who was to be such a thorn in his side a few years later, and Schönlein.

Middle-aged at the time Graefe was at the university, the first man to lecture in German instead of in Latin as he did in 1840 in his classes in medicine and therapeutics, the founder of modern clinical teaching in Germany and discoverer in 1837 of Schönlein's disease (peliosis rheumatica), Lucas Schönlein was to become in later years an eccentric. Regretted by not a single friend, he was to withdraw more and more from society, seeing patients only when it suited him and then treating them, his fellow doctors or whomever he chanced to meet, with the Göttliche Grobheit ("godlike coarseness") then considered fashionable decorum by more than a few in the medical profession. Once when a colleague reproached him for lack of respect and pointed to his own gray hair, Schönlein was supposed to have said, "Are you boasting? Even asses are gray." Graefe chafed under him, considered him a boor and a bounder but always maintained that he learned the specific knowledge of diagnosis from him.

In 1844 when Graefe started at the university, Rudolph Virchow, eight years older than himself, had not begun to teach there. Virchow was taken on at the Charitè as a prosector the following year, 1845, during which time he published 47 papers! and became full prosector in 1846. In 1847 he found the *Archiv für Pathologische Anatomie.* Two years later he was deprived of his prosectorship because of his outspoken democratic principles and left Berlin for Würzburg where he made a brilliant record as lecturer and teacher, so brilliant indeed that the sadder and wiser faculty could not rest until they had invited him back to the university. There he was installed as professor of pathology. A rather handsome bearded man, short-sighted and prissy, he was the first to observe and define leukocytosis, and between 1846 and 1856 created the conception of embolism, his greatest single achievement in pathology. A writer of medical history, biography, and the first to write upon medicine in relation to the fine arts (1861), he was resilient and sarcastic, and when it came along proudly opposed the Darwinian theory.

But there was more to university life then, as now, than studying, no matter how many great men presided. A week or two after school took up in the fall, the first dueling wounds began to appear on the young faces, freshly decorated with plaster, the bright eyes above them trying to seem indifferent. The majority of German university students in those days were noted for their pompous and insufferable manners, none more so than the surprisingly large percentage of fraternity members who had fought duels. The authorities pretended to frown on this practice but opinion was that fighting with swords would develop the warlike instinct so highly regarded by the reigning family and a half-chafing expostulation by his adviser was all the punishment that awaited the young duelist, while his satisfaction was immense, whether he won or lost. In a duel, each fighter, wearing a mask over eyes and forehead, tried to draw blood

from the cheek, chin or neck of his adversary. A wound on the cheek was the most fervently prized of all, the scar of which would remain through life as a testimony of its owner's courage, and if the wound was too slight the surgeon was requested to deepen it, unless this service was performed by the faithful "second."

Upon first observing these new wounds, and looking as a consequence more attentively at the pale scars on the faces of the older students, Graefe felt envious and longed for the day when he, too, would wear so sure a pledge of manliness. He mentioned his aspirations to his sisters Ottilie and Wanda, who, alarmed, banded with their cousin Mathilde and their mother, to prevent their being realized if possible. First, he was talked into forming his own fraternity rather than joining one of the existing chapters whose drinking, dueling, and rioting appealed so little to Graefe's female relations. It was not hard to persuade him to do this because he was a born leader and organizer and, a patrician, did not fall easily into the role of follower. Nonfraternity men were called Camels and Graefe, therefore, roguishly called his society *The Camellias.*

They met each Thursday and Saturday at Graefe's home, for bowling, singing, philosophizing, and drinking beer, a small group of boys who were to remain friends throughout life. Each member had a nickname. Graefe himself was called "Droll"; Frederick Schuft—later to have his name legally changed to Waldau, a young man of 22 at that time—was "Nero"; Arendt, "Kuemmel"; Michaelis, "Mug"; Oswald Frazer, "Knight"; Otto Roquetta, who later used a walking trip with Graefe (in a broad-brimmed hat hung with fern) as the theme for a novel, was called "Bembo."

Graefe studied, but he also sowed a few wild oats, memorizing and tenderly reciting Eichendorff's lyrical poems, lying at the feet of his sister's prettiest young friends on the grass at Haunt of Finches under the lighted Japanese lanterns that bobbed in the dark trees, or "walking and singing at night" when the moon was at the full and the moonlight whitest.

Sometimes he did not get home till the sun was coming up and would go to bed when all the world seemed full of the chattering of birds. He did not mind them. The military music was a different thing, though he had been accustomed to the sound since babyhood, and he used to press a feather pillow over his head and sink down as deeply as he could in another pillow, muttering under his breath, wishing the army and all things military at the bottom of the ocean.

He was not the only one who silently cursed when in the gray dawn light, the regiment that had stood guard at the palace during the night would go by, on its way to the parade ground for exercise or bound for somewhere else. In handsome infantry uniforms, helmets with spikes on top like the spikes on top of a big iron fence around a treasury building, with guns and bayonets bright as silver, the soldiers would stomp past in perfect step, with music accompaniment, fifes tootling and drums rattatting, cymbals clashing, and horns blowing, loud enough to wake the dead.

When they got past, Albrecht—and many another—would drift off to sleep again, only to wake with a start 15 minutes later when another regiment went by, with still louder music. And so it would go.

The comfort and convenience of all Germans, particularly in the vicinity of the large camps, was sacrificed to the army, and had been, for a century and a half, since the "good old days" of Frederick William I and his son Frederick the Great. Nobody dared protest. One-half or more of all the revenues levied in Germany was spent on the army or navy.

It was into this stern arena, this Sparta, that fervid, aberrant, and daedalian Graefe was born. He was a true romantic, but by physique he might have qualified for more than one rank and duty in the Prussian army where even the well born and privileged were

expected to be soldiers for quite some length of time.

How it was that in his whole life time, von Graefe should serve but from April 1, 1852, to April 1, 1853, as physician with the Second Guard Regiment, and then not on full duty, and with a good-natured company surgeon substituting for him in the ordinary routine, is not known. Perhaps his father's position at court, or his own great accomplishments even at that early age and at the beginning of his career, were what prevented his being forced to dissipate his talents in the officinal treadmill of military medicine. It was prevented, in any case—maybe by whatever it is that overlooks mankind's affairs and watches to make sure that the artist will come forth in his proper time and place with no interference, whether to build a cathedral, a ninth symphony, or that beautiful edifice, modern ophthalmology. Therefore Graefe served but one year in the army, working most of the time on eye patients.

What there is to be told of his military career might as well be recounted here. In early spring, 1853, the occasion of the *Frühlingsparade* came along, and thereby hangs a tale. . . .

Frühlingsparade was held at the parade ground near the Kreuzberg. This latter was a small sandhill, about 100 feet high, the most notable eminence in the vicinity of Berlin, with an iron obelisk on top. Here you could get a good glimpse of the city with the River Spree meandering through it, and see just south of it the parade ground where maneuvers were held. Periodically, the whole Berlin garrison was viewed by the Emperor, a custom handed down from the time of Frederick the Great (who was so stingy he blew his nose on pieces of old sheets rather than use good handkerchiefs). This review took place in the following manner:

With much pomp and ceremony all the soldiers were assembled. Then the Emperor rode up on a perfectly trained horse, so well trained, in fact, that if there had been movies in those days it could have got higher wages than Trigger does today. A very large staff of officers kept their mounted sovereign high-nosed company, and they and every other man on the field looked as if they had each swallowed a sword for breakfast, their backbones were that straight up and down. The king, Frederick William IV, rode along the lines and as he passed each regiment he said, "Guten morgen." The honored regiment instantly and joyously responded as with one ardent voice: "GUTEN MORGEN!" This accomplished, the different regiments went through their evolutions. The review ended with the troops leaving the field in the direction of the barracks and filing past the emperor as they did so, like a machine, rather than living breathing men.

Even young *Oberarzt* Graefe, with all his immunities, was commanded to put in an appearance at the *Frühlingsparade* in 1853, and so he did. But he was tardy, alas, and in a great hurry, wool-gathering, too, thinking of what he had been saying to Waldau about keratoconus only a few moments ago.

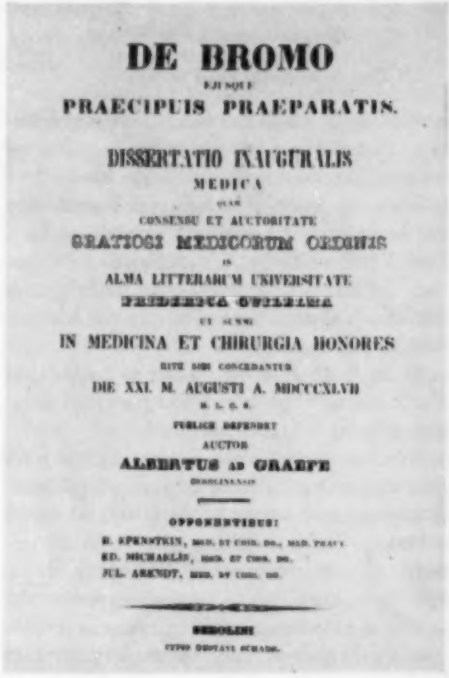
Contrary to the regulations of the guard corps, his long curly beard and long hair fluttered in the chilly wind. He had borrowed a uniform much too small for him—when it was buttoned across his chest, he couldn't take a deep breath or straighten up, so he walked stealthily, stooped over, having no idea what to do with the fine long hands coming out of the short cuffs except to clasp them behind him.

When the commanding officer spotted him, he said to his adjutant, "Um Himmelswillen was ist das? Get that lunatic with the beard off the field! For God's sake do it before his majesty catches sight of him! And listen," he added, "*keep* him off the field! I never want to see him anywhere near here again!" The commanding officer, though he pretended not to, well knew who "that lunatic with the long beard" was, and by this means saved the young scientist from such interruptions as this to his valuable work.

And that was the extent of Graefe's

career as a soldier for the fatherland, but in 1866 during the Prussian-Austrian war, he was in charge of the Municipal Cholera Hospital in Berlin. During this time he recognized that dehydration, the essential danger in cholera, could be combatted by injecting a sterile saline solution in the radial artery. This new treatment was significant and the paper he published on it received wide attention.

On August 21, 1847, he was graduated from the university, at the age of 19, receiving his degree at the hands of Johannes Müller. He wrote his 87-page thesis on bromide, and the principles of its preparations or forms, choosing this instead of the five other topics which interested him—"The more perfect the therapy, the fewer medicines," "Blindness is not a disease but a symptom," "Homeopathy acts merely through the curative powers of nature and faith," "The organism does not form new organisms," "The ideal form for the state is the republic—more practical, however, is the monarchy." His thesis was written in Latin, titled "De Bromo ejusque praecipuis praeparatis."


DE BROMO

EJUSQUE

PRAECIPUIS PRAEPARATIS.

DISSERTATIO INAUGURALIS

MEDICA

QUAM

CONSENSU ET AUCTORITATE

GRATIOSI MEDICORUM ORDINIS

IN

ALMA LITTERARUM UNIVERSITATE

FRIDERICIA GUILELMA

UT SUMMI

IN MEDICINA ET CHIRURGIA HONORES

RITE SIBI CONCEDANTUR

DIE XXI. M. AUGUSTI A. MDCCCXLVII

H. L. Q. C.

PUBLICE DEFENDET

AUCTOR

ALBERTUS AD GRAEFE

OPPONENTIBUS:

H. SPENATKIN, MED. ET CHIR. DR., MED. PRACT.

ED. MICHAELIS, MED. ET CHIR. DR.

JUL. ARENDT, MED. ET CHIR. DR.

BEROLINI

TYPIS OTTONIS SCHADII.

Fig. 4 (Ullman). *Dissertation thesis of 1847.* One of the results of the Revolution of 1848 was that such theses were, on the continent, no longer written in Latin.

Many a time had he discussed with his friends, the Camellias, the journey he was going to make when he was graduated, and now that day had arrived. His formal schooling was at an end. One more examination to pass, the Staatsexamen, a mere formality, and in December, 1847, he started his *Wanderjahre*. Not like the peregrinations of medieval days was this to be when ". . . boys went to study . . . the liberal arts at Paris, classics at Orleans, canon law at Bologna, medicine at Salerno, diabolics at Toledo, and good manners nowhere," nor like the Grand Tour for a young Englishman or American of Graefe's class and fortune, for the purpose of reading the "great book of mankind" in all its grandeur and folly. Our young aristocrat did not travel for experience and pleasure, or dilettante pursuits in art and language, either, but went in the humble search of medical knowledge, to the masters themselves, that he might learn at their feet. He did not go alone. His mother and younger sister Wanda, and his friend Waldau, went with him. They were accompanied by two servants.

Their first stop was at Prague, ancient capital of Bohemia, in the throes of dying as a German city and coming back to life as a Slavic, Czech one. A few weeks after the Graefe party had left the terraced town, a Slav congress was held there, the students had a battle with the soldiers of the garrison and the city was bombarded, but the trouble was soon over. From that time dates the rebirth of the Czech nation. Graefe and Waldau did not get in on any of this except the slight quiver and shake of the earth beforehand. Of course they felt the presentiment that Emily Dickinson defines as:

> . . . that long shadow on the lawn
> Indicative that suns go down,
> The notice to the startled grass
> That darkness is about to pass . . .

in the air. It made the Graefe women nervous, that feeling of something-is-going-to-happen, and they were glad when it was time to leave for Paris. The two young men were too busy studying with the ophthalmologist Carl Ferdinand von Arlt ("Papa" Arlt), as they did through January, 1848, and right up until St. Valentine's Day, to pay much attention to the rumbling.

If in Prague it was *going* to happen, in Paris it was happening! They arrived there smack bang into a revolution!

Machinery and other new processes were now beginning to come into use, large-scale industries had begun to grow up, economic centralization was taking place, and the insurrection in progress in February, 1848, may have been due to such phenomena.

It was bloodless at the beginning, a revolution to abolish poverty by the organization of labor, but for all its tranquil look, it was a power irresistible enough to spread, thanks to improved means of transit and communication and the great development of the public press, like wildfire to all the civilized communities in the world except Switzerland, Great Britain, and the United States, where alone democratic principles prevailed. It was caused, as might be expected, by more than one grievance.

The National Guards were called out but as they marched here and there, their cry, too, was "Vive la réforme!" so it is not surprising that when the populace at large and the regular troops fought, the guards jumped in on the side of the people. That being the case, officers and soldiers thought twice before they assaulted their fellow citizens and were reduced by this unsoldierly prudence to inaction. Thus, and almost without bloodshed, the insurrection triumphed. But there was more to come, and soon. Paris was not to live "happily ever after."

Paris! Peter the Great once said, "If I had a city like this, I would burn it to the ground!" When asked in astonishment why, he answered, "For fear it would absorb all the rest of the empire!"

Graefe's heart began to beat faster when he saw it. The branches were still bare, dirty snow was shoveled to either side of the walks, the February air was icy cold, but something about its beauty and vitality warmed his blood. But young Albrecht von Graefe, with a program of work laid out that would choke a horse, was not really going to have time to enjoy the imponderable city. He was not there as Thackeray was, and at the same time,

> With a wild mistress, a staunch friend or two,
> And a light heart still breaking into song:
> Making a mock of life, and all its cares,—

but for sober study. If he had known, then, that 20 years later, more or less, everything would be over for him, would he have—? But these are idle speculations. Art is long, in truth, as the saying goes, but life in youth feels as if it would outlast it by many a blue moon.

"I limit myself to the important men," Graefe wrote a school friend, "and study their methods. . . ."

Armand Trousseau, internist at the Hôpital St. Antoine, was one of these. A composed man with wavy pompadoured hair, he was a master of clinical delineation. He had written a treatise on laryngeal phthisis impressive enough to win a prize from the Academy of Medicine, and was the first to do a tracheotomy in Paris. Graefe enjoyed watching and listening to him, but he wrote, "Internal medicine here is mostly a loss of time. Diagnosis is not as exact as in Prague and therapy does not seem to be as judicious as in Berlin. I never saw so many students, all making aimless declarations. Lectures, somehow, leave one unsatisfied. I don't bother with the clinics of Bruillard, Chanel, or Rostan after a sample of their wares. Pieny is as mad as a hatter. I wouldn't mind Louis, only that he is so hoarse he hardly ever opens his mouth. Actually, I spend

only a couple of hours with internal medicine—early in the morning, to get it over with.

"Surgery," he went on, "is a little better. The Frenchmen operate fast and well, but they put bandages on like wounded soldiers bandaging one another in the midst of battle. There are lots of surgical patients, to be diagnosed by sight or touch, but they lie in 20 different hospitals, all over town, on both sides of the river. One would have to travel miles to see them. As a consequence, I see about as many patients of this type as we saw at home. Malgaigne has cast a spell over me. I never heard such an interesting lecturer on operative and experimental surgery, fractures, and dislocations. He edited the works and wrote a wonderful biography of Ambroise Paré. I like the way his mind works—you know he invented some most ingenuous hooks with which to treat a fracture of the patella. Nothing but a *very* rare case that all the students are buzzing about, or the announcement of some really big operation in one of the amphitheatres, can make me forego one of his lectures."

Claude Bernard, successful playwright turned physician, 35 years old in the year 1848, was another man Graefe respected. As J. B. Dumas said, "Ce n'est pas un grand physiologiste: c'est la physiologie même." And so he was, not so much a physiologist as physiology itself. He preached that the chief aim of physiologic experimentation is to throw light upon morbid conditions and can be said to be the founder of experimental medicine. "He is investigating the glycogenic function of the liver, a project he stumbled onto by chance," Graefe wrote, "and will publish some interesting papers. He says such stunning things. I must try to remember some of them. He resides where he does because that happens to be the district of a certain police-commissioner with whom he accidentally struck up a friendship. And? you ask. What is the connection? Why, because this police-commissioner makes him practically immune to persecution as a 'vivisector of animals.' I understand he even has trouble with his wife and daughters on this score, until he is in despair. . . . He is taller than I am and looks just a little like the composer of that singular opera Tannhäuser. . . .

"I didn't intend to study much dermatology here, as I was saving that for Vienna and the clinic of irrepressible von Hebra, but I am swallowed up by Ricord. What a Corypheus that man is! I have to keep tight rein on myself or he would have me believe that syphilis is the most interesting disease in the world. When he talks about it, it is, I swear. You know it's named after a legendary shepherd. . . . Ricord is middle-aged but well-preserved. He looks as if he had intended to laugh, but decided not to just when his face was all prepared. They say he is the most popular man in Paris and I don't wonder. There never was a more attractive personality—he draws people like moths to a flame. He dresses like a prince, is supposed to live in absolute splendor, and the report is that he spends a cool 100,000 ducats a year. Well, you should see him—he never walks but runs—and hear him—he always seems to be joking. You would double over to see him with his patients—they worship the ground he walks—pardon, gallops—on. Once in a while when making rounds, he'll forget some patient and the fellow will call out 'Ricord!' indignantly—just like that, Ricord! not doctor, professor, even monsieur—and back will trot the American—it is said he was born in America, of a French mother and father—to the bedside, with some sally on his lips that will make the neglected one, plus the rest of the patients in the ward, even if some are dying, roar with laughter. I never knew a man could be so witty or so completely dégagé. It is a revelation. . . . You know, in Berlin nobody knows anything about syphilis. . . .

"If you want specialties," he wrote a few days later, "come to Paris, where they 'bloom twice.' Above all the rest I place ophthalmology. I attend two clinics—Sichel's and

Demarres'. You can't imagine what a fiend for work that Sichel is, or the amount of material in his clinic—30 to 40 new and 300 to 400 old patients a day—or how insupportably monotonous he is. He drones on and on like a hive of bees until the first thing you know your head falls down on your chest with a jerk—and you discover you have been asleep, almost breaking your neck. . . . I watch about two cataract operations there a week."

Sichel's clinic for diseases of the eye was located south of the Seine at the Rue Jacob. He had the sunless courtyard and the back half of an old dwelling, the front part of which housed a mattress factory.

Louis Auguste Demarres started out as a tax collector but he could also give violin lessons and paint well enough to earn a little money from the pictures. He began to study medicine at an early age. It was when he was working as a forester, however, that he had the honor to make the acquaintance of Sichel who owned some property adjacent to the small state-owned woods the young man was taking care of. Sichel was in some embarrassment—he had lost the key to his iron gate, but luckily the forester had found it. . . . Some said, in later years, that the key Sichel had let slip through his fingers and Demarres had picked up, was the key to ophthalmology. It was a case of finders keepers, losers weepers! The young forester on that historic occasion knew the man who was addressing him. Demarres had often attended his lectures, he said, and Sichel thought he had noticed him at the clinic but wouldn't swear to it. In 1839 Demarres became the older man's assistant, leaving him in 1842 to branch out for himself.

"Demarres," Graefe wrote, "has much less material—6 to 8 new and 50 to 60 old patients a day—but I don't care about that. He used to be Sichel's pupil, but thank God didn't take lessons on how to lecture from him. He's wonderfully keen and forcible, never dull for an instant. His clinic is lots more comfortable than Sichel's, too, where everybody has to stand all day long. You can sit down, if you want to, patients as well as physicians. That can be quite pleasant, you know, after being up since daylight. I like to be there. . . . You ought to see Demarres operate, sometimes 6 operations in a day. Some of his wizardry I don't approve of, though, and he is none too gentle with his patients. You ought to see him evert the lids or probe the tear duct—one thing you can learn from him if nothing else and that is *the bold push*. You learn to take the bull by the horns! On his right forefinger when operating, he wears a big ugly ring* which he invented and had especially made for the purpose. It's got two devilishly sharp tines or barbs sticking out of it, one on either side. They're to impale the sclera with, while he couches, his instrument held between thumb and third finger. . . . I do quite a bit of operating. Demarres goes home early, leaving the stragglers to us. On the whole I don't do too badly, although yesterday somebody bumped my chair just as I had my knife in the eye of a poor nearly-bald female creature, I pierced the iris, and there was a lot of bleeding. I felt very remorseful. . . .

"It's the finest warm weather after the chilly spring. The grass is green, the trees shady, and I don't know which are prettier, the flowers in the parks or the girls. But all I can do is smell the former and give a sidelong glance to the latter—or vice versa—as I hurry by. I spend quite a lot of time with the microscope and have collected 52 specimens of cataracts. . . .

"My mother and sister visit, go shopping in, sightseeing and in general enjoy the different cities of Paris, except the scientific one, which they leave to Waldau and me. They have, by the way, a musician in tow.

* In New York City in the 1880's, Dandy Johnny Dolan, a member of the Whyos gang, also devised a ring with which to operate on eyes. According to Herbert Asbury, in *The Gangs of New York*, Dandy Johnny "improved the technique of gouging out eyes; he is said to have invented an apparatus, made of copper and worn on the thumb, which performed this important office with neatness and dispatch."

. . . Spontini, the Italian composer, an old man now, like an old eagle, with eyes that still shoot out sparks."

The cold winter passed uneventfully. Graefe was more at Claude Bernard's institute than anywhere else, studying the action of the oblique muscles of the eye in rabbits and dogs, the subject of his first notable paper. He was working even harder now than previously, trying to see more and do more, and his mother said solemnly that the attack of illness in February that laid him low for several days—it turned out to be a rather bad case of measles—was a warning to him to rest on his oars for a little. In Paris! she said, and slaving like an articled clerk, either absent altogether, transcribing notes or looking himself blind in that odious microscope; having to be dragged to the theatre or a concert—and he had to admit it, he had been to only one large ball! and what happened there? Why, he stayed in the company of that boisterous man Ricord almost the entire time. Really, she wondered what the young men were coming to nowadays. Paris. . . . Spring in the air. . . . And doing nothing but study.

One Sunday in April, 1849, Graefe and Waldau paid a call on Heinrich Heine. He was in bed where he had lain since the previous spring, in the "mattress grave" that was to confine him, a victim of paralysis, for another seven years. It was in this poorly furnished and not clean chamber that some of his most radiant work was to be composed, but neither he nor his visitors could guess that.

To his young countrymen he seemed an old man whose race was run. He was 51. Only his eyes were fine still and they looked weary. He said he was engaged in a course of reading. He was reading all the medical books he could find that dealt with his sickness. "I don't know what good it will do me," he said ruefully. "Except I will be an expert. I can give lectures in heaven, perhaps, on how much the doctors don't know on diseases of the spinal marrow!"

When he learned that the next year Graefe was going to London for a few months' study, he looked interested. "I have been there," he said. "And many times since, I have thought that I would like to live there if it weren't for two considerations. . . ."

"The climate," Waldau ventured, "and—?"

"The coal-smoke," Heine said gravely, ignoring him, "and Englishmen. I can't endure either!"

Before they took their leave, Frau Heine, the ignorant, vain, good-humored creature the poet had married eight years before on the eve of a duel he did not expect to survive (she had been his mistress for nearly a decade at the time, and before that had sold boots in a shop), offered them some wine, and they drank to the sick man's speedy recovery.

Graefe had to remind himself over and over in those mean surroundings that this was the great lyric poet, who wrote "Die Harzreise" and "Buch der Lieder," that this was the wit and satirist, the Aristophanic genius known from one end of Europe to the other. It did not seem possible.

> Ich weiss nicht was soll es bedeuten,
> Dass ich so traurig bin. . . .

After a stay of 20 months, Graefe left Paris for Vienna. He continued his *Wanderjahre* alone, the journey having come to an end for his mother, sister, and Waldau who returned to Berlin.

Austria was busy, as she had been for some time, gaining a free hand in her Hungarian and Italian dominions—in other words, engaged in war (it was Graefe's lifelong fate to live, study, travel, and work against a panorama of war that burned everredly like a sunset about him).

It was with curiosity and anticipation, not unmixed with awe, that Graefe entered the city where Marcus Aurelius was supposed to have died, where Charlemagne hung his hat at least long enough to put in the foundation of St. Peter's Church, where lilacs *first* in the dooryard bloomed (brought from the

Orient and planted there before they were known anywhere else in Europe), and where the idiomorphic Viennese cafes with their 50 kinds of coffee owed their existence, at least indirectly, to a Turkish siege in the 17th century and one of its consequences, the Kipfel, conceived by a genius baker in memory of the great day of victory (when against the swarming Viennese the Moslem scimitars might as well have been made out of cookie dough). And Vienna came up to all his expectations.

It was raining hard when he arrived, but it was a warm straight-falling September rain that only made the scene look fresher and brighter, enhancing even the fortifications, later to be razed to make way for the Ringstrasse, enclosing the Inner Town, where he was to stay in a hotel on the Graben.

The medical faculty and the facilities for teaching at the University, an institution then nearly 500 years old, were in the very front rank at that time. Graefe planned to listen to Carl Rokitansky, the merry pathologist who was to do 30,000 post mortems in some 50 years, Alois Bednar, tutelary saint of children; Ernst von Brücke, from Berlin, now occupying the chair of physiology in Vienna; and met again with much satisfaction and warm rapprochement, Johannes von Oppolzer, inspired diagnostician, sharp-tongued Ferinand von Hebra, informal psychologist and official dermatologist, whose clinic was as popular in Vienna as Ricord's was in Paris, and others. But it was obliging Eduard Jäger and his father Ferdinand Jäger, a rather frail old man in semiretirement, who took up most of his time and attention.

The Jäger ophthalmic clinic was situated close to a military hospital, the Josephinum Academy. A majority of the patients were soldiers, their wives, and families, and many of the students were preparing to become military surgeons. It did not take the Jägers long to perceive that Graefe, who won their hearts from the beginning, was a young man of unusual capacities and they let him make himself at home, to use their clinical and private patients as he would. Four years later, Eduard was to dedicate a book, "Ueber Star und Star-Operation" to him.

For the first time in his life, Graefe now essayed to lecture, displaying at the outset much of the flair, finesse, and yet artlessness, the emotion and skill, that were to make him a world-renowned lecturer and teacher. His initial audience, who had come to him in a body some days before and begged him to talk to them on the theory of diseases of the eye, was a group of young doctors from several different countries engaged in the study of ophthalmology. Like the Syriac lady's irresistible stories, one lecture led to another. He held his listeners in the palm of his hand.

Once he was asked, "Will you speak to us on the treatment of the eye?"

"I can't," he said with regret. "I don't know enough. But come to Berlin and we'll see if we can't learn something about it together."

The long icy winter, his third away from home, seemed in retrospect to vanish as suddenly as the last fitful fall of snow in April. Soon the weather was so hot that in the ophthalmic clinic slippery eyelids could with difficulty be spread by slippery fingers, patients' perspiration-wet heads with slick hair were as hard to hold as a greased pig, and flies, coming in clouds through the open windows, clung to everything.

Seeking a breath of fresh air one evening after a scorching day Graefe strolled out with old Dr. Jäger. In front of the Hofburggate, to his surprise he saw a face out of his boyhood, that of a friend, Jean Renaud. The two former schoolmates after exuberant greetings and a spate of questions, exchanged news of their activities past and to come, while the old doctor smoked his pipe and gazed reflectively at the stars.

Son of a bookseller, Jean had taken up his father's trade with great success. His uncommon sedulity, often perceived in those who will die young (his tombstone would give his age as 26) and later remembered

with astonishment, was on the eve of being handsomely rewarded, for his proud parent had purchased for him a small publishing house in Berlin. He expected to go there at the end of the month. "It is a coincidence that I meet you," he said, "especially since you say you are going to be an eye physician! You see, the first thing I am going to publish will deal with the eyes! A professor of physiology in Königsberg has written a little paper about something called the eye mirror and I am going to publish it."

"Eye mirror?" Graefe asked.

Old Dr. Jäger took his pipe out of his mouth and cocked an ear toward young Mr. Renaud, waiting.

"That's what he calls it. It's a thing to look at the sensitive membrane of the eye and the optic nerve with—for the first time in the eyes of breathing men, so he says."

"Who is this professor of physiology?"

"He's a friend of my father. His name is Hermann Helmholtz."

In the next few days, the invention of Helmholtz was so constantly discussed and speculated upon that, if there was anything to the old superstition, the professor's ears must have burned to a crisp.

Ernst von Brücke had heard rumors that an instrument for examining the retina of the living eye had been constructed and this must be it! He himself had tried to devise a tool of the same kind but could not bring in adequate light. His hope was, that he would get deflective and refractive illumination effects by means of a hollow glass placed at an inclined angle over the eye under scrutiny, but this had not materialized. Now such an instrument had actually been invented! and a paper telling all about it was soon to be published. What vistas might not this advent open to ophthalmology.

It was with impatience that the young and old doctors Jäger, von Brücke, and Graefe settled themselves to wait the dispensation from heaven—as they thought it, and as it was to prove—Professor Helmholtz's new "eye mirror."

In July, von Graefe bade goodbye to his teachers, friends, and the city of which he had grown so fond, and left Vienna for Prague. There he spent two weeks with good old Papa Arlt, who accused him of growing half a foot in height and never stopped asking about Parisian and Viennese doctors, medicine, medical publications, procedures, and lines of conduct, Graefe doing his best to satisfy the boyish curiosity he could not but admire. It was at this time that he learned a valuable trick in teaching surgery of the eye.

"When I taught my assistants," Carl Ferdinand von Arlt wrote in his autobiography, "I made it a strict rule never to interrupt them when I saw them making a mistake but let them finish first. Then—and then only—did I explain what they had done wrong. Later on . . . when I went to Berlin and saw Graefe in his clinic, he told me he used that same method and that he felt greatly obliged to me for it. . . ."

It was a happy day for the Graefe family when, the last week in July, their wanderer came home, wearing maturity like a new coat and looking even handsomer than they remembered. But he was only going to stay a little while, he said, having still to visit England, Scotland, and Ireland.

"You will be an old man with a long gray beard by the time you get through traveling," Ottilie teased her brother. "You are like the tribes of Israel. When will you come to rest?"

"I shall be back by the middle of October."

"This year?"

"This year. And then you will see something happen!"

"What?" Wanda pleaded.

"You must wait and see."

Albrecht and his mother, to whom he had already written of his final resolve and settled purpose, to open an ophthalmic clinic in Berlin, exchanged beaming, mysterious glances.

Graefe arrived in London at about the time the Kohinoor, originally an 800-carat

diamond reduced to 270 carats by the botching job done on it by frightened European diamond cutters, was being presented to young Queen Victoria.

In the hackney coach on the way to Moorfield's Hospital, gazing with interest to either side, he wondered idly where the Romans built their bridge (that was supposed to have got this great city of London off to a start) across the Thames. Londinium, they had called it. In August weather, close and hot, it was not a very attractive place, that was certain, lying under a thick layer of moving smoke or fog through which sunshine crept down in dim rays as yellow as old paper. It seemed noisier, dirtier, bigger, and busier than any other city in the world, as full as Constantinople with beggars and cripples. By night cutthroats and footpads were said to prowl the streets where still worse scourges lurked at all hours. A liveried footman's sore throat led to the discovery of 50 brimming cesspools under Windsor Castle itself, so there was some small reason for infection and disease in the town said to be 20 centuries old.

Moorfields, in Bloomfield Street, the first hospital in England dedicated to eye diseases, was opened in 1804 as the Royal London Ophthalmic Hospital. Forty-six years later, there were five such institutions, but Moorfields, with 30 beds, was the largest, and the best known; 8,000 patients came there for treatment a year. With lively interest Graefe looked forward to meeting the two chief doctors, Bowman and Critchett, and he liked them both.

William Bowman, as small and thin as a jockey, was a famous microscopist at the age of 25. Then he turned physiologist and after that became an ophthalmologist.

Light-fingered as the most talented pickpocket, nerveless and tireless, an original thinker, he had been chief of Moorfield's eye clinic for four years. It was at Joldwynds, Bowman's country estate in Dorking, that the young German visitor was invited to stay during his London visit and from which green and delectable spot the guest and host repaired each morning for London. At Joldwynds, scenes of almost exaggerated Victorian domestic bliss were enacted in the evening between the virtuous father, pious mother, and large family of classical children against the ornate gaslighted background. Wife and family, however, were not in evidence on the two or three occasions that large dinners were given in Graefe's honor.

At the first of these, three of the guests among 20 were Sir James Paget, London's most famous surgeon, but a few short years removed from poverty and privation; eccentric Dr. James Wardrop, then 69 years old, who might not have retired permanently when he did, at just past 40, if he had realized his retirement was to endure for nearly half a century; and Dr. George Critchett, who worked with Bowman at Moorfield's.

Indefatigable, humdrum, of the artist breed but lacking art, Critchett held his cataract knife artistically, cogitated paintably, and lived picturesquely. He would rather talk sculpture than surgery, and music rather than medicine, but he was a conscientious physician.

The young man on his left who was perfectly familiar with his paper on "Fungus hematodes of the retina," published more than a decade before the death of George III, interested sulky old James Wardrop. The word ophthalmology was not then in current use, nor ophthalmologist. "Bowman tells me you are going to do eye work," he said crossly.

"Yes, Sir," Graefe said, smiling.

"Most unsatisfactory, young man."

"May I ask why, Sir?"

"Why? Why, because it is not recognized as worthy of a full professorate, that's why, or one of the reasons why. And the nonsense! Tell me. Would you operate on a cataract when the weather was stormy, hey? When it looks like rain?"

Graefe laughed.

"It was so tantalizing. There was so much

to do and nothing to do it with," James Wardrop said.

"Was, Sir?"

"No way to measure properly! No possibility to look into the living human eye! Dolts at work at one's elbow! Idiots expressing their opinions! Jealousy! Stupidity! And palaeoliths to work with, tools for prehumans. . . ." The old man shot an angry glance at his listener. "I laid them down twenty-nine years ago and it might have been but yesterday. Not a single new instrument worth taking in your hand, has come . . . there has been no advancement whatsoever. . . ."

"Professor Helmholtz in Königsberg has invented an eye mirror which is said to —"

"Another palaeolith, be sure. Tools for cave men!"

Graefe was silent, not wishing to dispute.

"I am bitter, you think. And so I am. And so is the lover of the Syrenic voice belonging to half a woman, half a fish! But listen to me, young man. You will discover your beautiful specialty to be the most teasing, inconstant, bewildering, contrary, and entirely wonderful thing a man ever tried to get hold of, only to have it wiggle out of his grasp and glide away beneath mystery's deep blue ocean. . . ."

Promising to return the next year for the Great Exhibition, Graefe bid his London friends goodbye and travelled to Glasgow. On his way to 72 Charlotte Street, the Eye Infirmary of William MacKenzie, he heard the chimes from square-towered old Tolbooth and remarked the unusually tall chimneys pouring out clouds of black smoke that smudged all the buildings, obscured whole streets and gave even clean and prosperous citizens a grimy look.

The author of a then celebrated textbook of ophthalmology, William MacKenzie proved an amiable host, but Graefe, shuddering at the thought of the haggis prepared especially for him, was glad to discover after a day and a night that there was no real reason to stay in the Scotch city and that he might as well go on to Ireland.

The land of kings, 171 of them altogether in its long history, lay green under the soft sky. Four years earlier, the potato crop had failed, there was a famine, and people, whole families at a time, had starved to death. During 24 hours, in one district alone, 11 dozen dead bodies were found along the road. Then came a contagious fever. Between the two onslaughts, thousands upon thousands perished. But there was nothing, though so recently enacted, to remind the casual visitor of tragedy.

Graefe was not aware that for every three persons he saw, most of them smiling, in the Irish way—rather, for every three persons alive in Ireland at that time—a corpse lay under the ground, dead of plague or starvation and buried during the winter of 1846-47.

Sir William R. H. Wilde, of whom Shaw once observed that he, "like Frederick the Great, was beyond soap and water, as his Nietzschean son was beyond good and evil," was 35 years old at the time of Graefe's visit in September, 1850. Garnished to the gills and smelling like a flower garden, he was single and would stay so, the delight of Dublin society, for another five years until snared by lovely "Speranza," a union with whom was to bring forth none other than Oscar himself, all in due time.

Sir William lived at No. 15 Westland Row, a good 18th century house with a shiny brass door knocker and pots of geraniums along the sills, presided over by his mother and sister Margaret, where he carried on a large medical practice. He was really an otologist (and a discoverer of prehistoric lake dwellings on the Irish crannogs) but eye work interested him, too, and he had so notable a reputation as an ophthalmologist that Graefe would not have thought his *wanderjahre* complete if he had not come to see the famous surgeon at work. And indeed his teachings proved invaluable to Graefe, who stayed in Dublin more than a week.

Step by step, with scrupulous care, the captivating Irishman showed, by what he did, *what not under any circumstances to do,* and Graefe paid close attention to these backhanded instructions. Sir William apologized for the lack of a proper hospital. "I'm going to establish one someday," he said, "along the order of Moorfields."

His great passion that early autumn was a new toy, a Buchanan's lamp, constructed, not unlike a magic lantern, of a tin box with a candle inside and powerful reflector, across from which was a tube containing two covered lenses. These threw a disk of light about the size of a quarter on whichever organ was to be examined, and Sir William said its invention was comparable in scope to that of the steam engine.

He claimed that 85 percent of the 120 cases he had operated on for crossed eyes in six years were successful. But Graefe thought otherwise of those that came under his scrutiny. The Irishman proudly went through his paces and performed several operations for strabismus in front of Graefe, who bit his lip and tried not to wince at the demonstrations, skillful as they were and lightning like as Sir William worked upon his unanesthetized patients, severing the tendon of the internal rectus muscle. The results were that thereafter, the eyes that used to turn in, turned out, and vice versa.

One of these lucky fellows, who had been accustomed to gaze at nothing but the bridge of his nose but now could look up the street and down the street at the same time out of the corners of his eyes, was an agreeable if somewhat weird bachelor named Mr. Shaw. A few years later he would marry and beget a son, George Bernard by name, but it's a wise man who knows with whom he is being asked to shake hands, and nothing about Mr. Shaw gave away the secret that he would one day have some small connection with fame, extinct though he might be at the time.

On Sunday, Sir William carried his guest for a drive through Phoenix Park, where a hundred duels were fought every year, and to St. Patrick's, the great cathedral where once had stood the little church Ireland's true saint put up with his own hands, or so the story went, after he shooed the snakes away forever and before he died in the year 495. (A fellow passenger on the small boat crossing the North Channel inquired of Graefe if he had seen any serpents while on the Emerald Isle. "No, and you won't," the man said. "Sometimes they bring them in for money, you know, show people and such, but the creatures always curl up and die after a few days, for a snake don't dare to live in Ireland, that's the God's truth!")

When Graefe departed, Sir William's mother was rather severe for a few days with her apologetic daughter Margaret, who seemed doomed to spinsterhood, while the young maiden herself wondered wistfully why such a handsome and rich young man would want to devote all his waking hours to learning to be an eye specialist! Surely, there wasn't that much to it, to master. Surely, he'd have some time for a little harmless recreation. After all, according to William, he had been studying from one end of Europe to the other for three long years, and before that, had been a scholar for four years at the university of Berlin. When was he going to understand that a pretty young lady might be smiling at him for a change? And when was he going to live?

In an October gale tearing off the upper snow of the Monte Rosa mass and hurling the frozen spray in their faces like a shower of needles, Graefe and Waldau, bound together with ropes, made their way up the Zumsteinspitze summit. Halfway up the mountain, they rested beside a small shelter out of the wind, resuming the conversation that had occupied most of the previous day and evening down below them in the town of Zermatt. Why they had left the rosy confines of the luxurious public house with its lamps, its fires, for the peril, punishment, and hard labor of mountain-climbing, those

men of mettle know best who would do the same. They had started off before daylight. Now at mid-morning they had halted and were taking a moment's respite, anchoring their alpenstocks, that otherwise would have been as slippery to hold as eels in their ice-coated gloved fingers, in snow, and leaning upon them.

"And that is why I think it will be the best hospital for eye diseases in the whole world," Graefe continued. "Can you see any drawback to the plan?"

Waldau shook his head. "Only that you might need a few more hands and feet before you get done," he said. "Just one pair of each will run you rather short, won't it?" He smiled teasingly but as he gazed at the forbidding landscape his face grew sober.

"Not with you to help me," Graefe said. "I'm not worried, and I know you're not either." He too then turned reflective as he looked over his shoulder and upward into the blinding glitter of the way they would go. "You know," he murmured after a long silence, "I wonder if life itself is not like these mountains . . . sublime, but with a capability for cruelty that makes a man's blood freeze in his veins to think about it. No mercy, none whatever. One false step—if your head swims for an instant, if your hand falters, if your heart quails—down you hurtle to be torn to pieces on the jagged tusks of the earth beneath as though you had been flung into a den of wild beasts!"

"The awful part of it, to me," Waldau said softly, "is when in life a man does *not* make one false step, when his head does *not* swim, or his hand shake . . . when his heart does *not* fail, and yet he is hurled down sooner or later to the same fate! and takes the same plunge, the same mauling. Drivellers and oracles alike, cowards and heroes."

A cloud passed over the sun, obscuring it, bringing grayness across the bright morning. The two climbers shivered at the same moment, and Graefe turned his collar up higher.

"I'd rather suppose a man deserves his destiny," he said, loosening his alpenstock from the packed snow. "Otherwise how could we reconcile our good lives with our consciences? For what have you and I done to deserve all our joys—and the paramount one to me is to have found my work and to know I am going to be able to pursue it without interference how and where I will —more than other men? No, no, my dear Nero. Let us comfort ourselves! Let us pretend we deserve everything that happens to us, bad as well as good. That way, there is less accounting to do!"

The sun shone out now and they started on up the dazzling mountain, while the wind surged against the cliffs beneath them with a full deep boom like the ocean.

(To be continued)

Ophthalmic Miniature

And there was again war at Gath, where there was a man of great stature, who had six fingers on each hand, and six toes on each foot, twenty-four in number; and he also was descended from the giants.

The Second Book of Samuel,
Chapter 21, verse 20.

Perhaps the first mention of the Laurence-Moon-Biedl syndrome.

REPAIR OF SEVERE ENTROPION OF THE UPPER LID

A CASE REPORT

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New York

The surgical treatment of entropion of the upper lid is currently the source of considerable interest and numerous procedures have been devised for the correction of this condition. Results are usually satisfactory when the loss of tarsus and conjunctiva are of minor extent. When the entire width of tarsus has been lost, along with the conjunctiva covering it, repair is a difficult problem. Techniques employed in partial width losses fail in these cases, and the lid appears only to turn farther inward with scar contraction.

The following case history illustrates a method of repair that provided a good cosmetic and functional result.

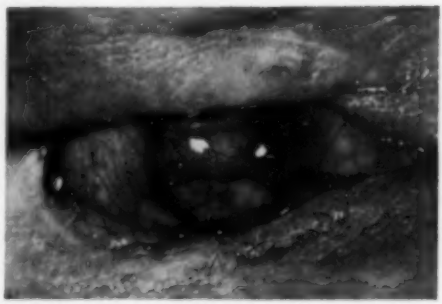


Fig. 1 (Cole). Severe entropion resulting from reconstruction of upper lid by pedicle graft following burn. Minor entropion present in lower lid.

CASE REPORT

The patient, J. C., was injured in factory work when a hot ingot fell on him burning the entire side of his face. The wound was closed with dermatome and pedicle grafts. When healing had taken place, the remaining defect consisted of loss of the outer half of the right brow and upper lid and eversion of the lower lid. The cornea was opaque.

Reconstruction of the lid was done by means of a tube pedicle graft from the neck. Following this repair, the patient complained of difficulty in completely closing the eye. Slight entropion of the lower lid was present and the upper lid, being formed of skin both inside and outside did not furnish adequate lubrication. Several fine hairs irritated the sclera.

An attempt was made to replace the skin lining of the lid with conjunctiva by using lower lid tarsal-conjunctival layer. This repair was unsuccessful in relining the lid and

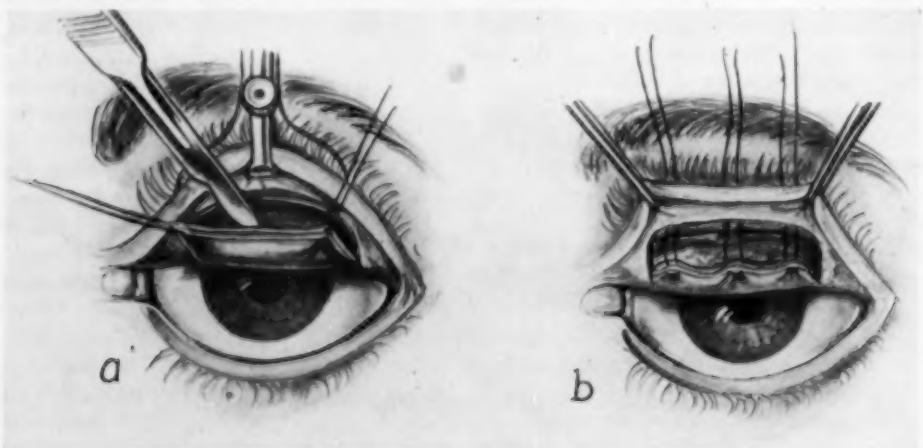


Fig. 2 (Cole). Method of taking tarsoconjunctival graft from opposite eye.

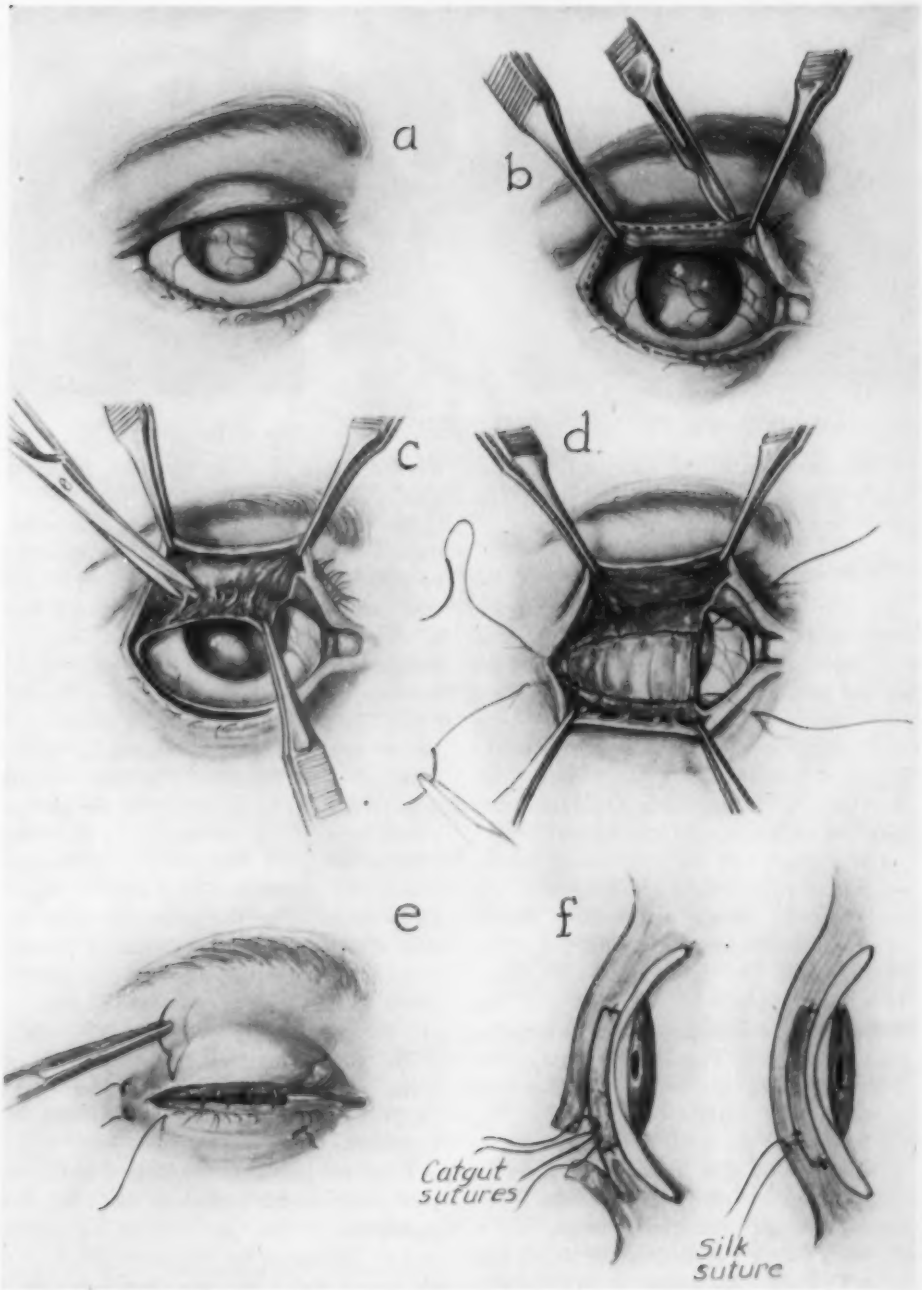


Fig. 3 (Cole). Technique of implanting graft to repair entropion of both lids.

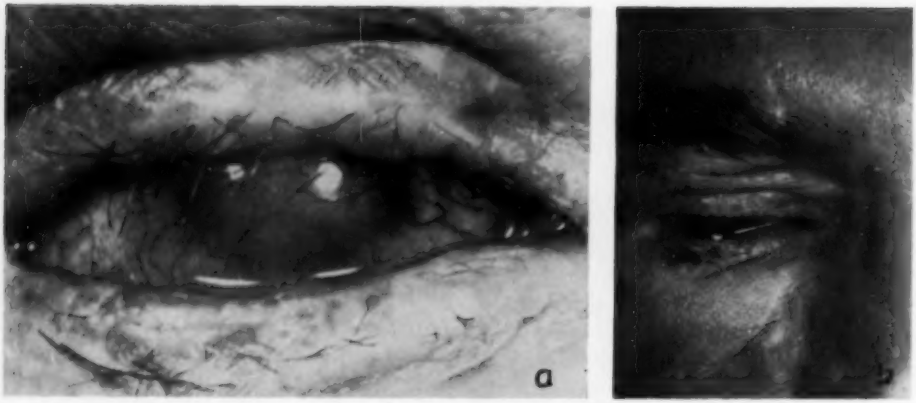


Fig. 4 (Cole). End result: (a) 12 weeks postoperatively, (b) Six months postoperatively.

created entropion of the lower lid. The patient was first seen by me at this stage. The condition is shown in Figure 1.

PROCEDURE

In entropion of minor degree, narrow, wedge-shaped tarsoconjunctival grafts have been used to lengthen the inner layer of the lid and turn the eyelashes outward. These small, free grafts take very well, since the blood supply is abundant in this area. It was therefore thought that a larger free graft from the opposite eye might be useful in this case. The technique used is as follows:

The upper lid of the good eye is everted and a tarsoconjunctival graft taken in the usual manner, leaving a three-mm. rim of tarsus and conjunctiva at the margin of the lid (fig. 2-a). The defect is closed by mattress sutures extending from the upper edge of the levator-conjunctival layer through the edge of the tarsal rim and out through the skin where they are tied over rubber pegs or buttons (fig. 2-b).

The bed for the graft is prepared on the opposite eye by first incising the upper lid margin along the entire length of the defect and continuing this incision along the edge of the lower lid for a corresponding length (fig. 3-b). The skin-muscle layer is then dissected from the conjunctiva of the upper lid (fig. 3-c). The lower lid skin and muscle layer is separated from the tarsoconjunctival layer for a distance of *about* two mm. from the edge.

The tarsoconjunctival graft is attached to the conjunctiva and levator of the upper lid by a continuous silk suture which begins and ends on the outside of the lid so that it may be removed later. A similar continuous suture attaches the lower edge of the graft to the tarsoconjunctival layer of the lower lid (fig. 3-d). The procedure from this point on is similar to the Hughes operation for reconstruction of the lower lid. The lower lid skin is sutured to the tarsal surface of the graft at a level a millimeter or two above the lower edge of the graft with interrupted chromic catgut (6-0) sutures. The skin of the upper lid is anchored to the graft and lower lid skin at adjacent points with catgut sutures. The skin of the upper lid is anchored to the lower lid skin with interrupted silk (fig. 3-e and f).

The continuous and interrupted silk sutures are withdrawn in five to seven days. In 12 weeks, the graft is divided and lids separated.

Excellent results were obtained in the one case in which this procedure was tried. The postoperative result is shown in Figures 4-a and b. The entropion was corrected in both the upper and lower lids. Eye lashes were restored by the usual method and corneal grafting is being considered for the future.

780 Park Avenue (21).

MAXIMUM AND MINIMUM RESECTION OR RECESSION*

Results of a five-year study

Beulah Cushman, M.D., and Robert L. Willard, M.D.
Chicago, Illinois

We wish to present the results of the analysis of 137 squint cases, collected over a period of five years. In all of these cases a definite standard of values was followed which we hereafter refer to as the maximum and minimum amounts of resection or recession.

In 1947, Dr. W. F. Johnson of the New York Eye and Ear Infirmary intended to publish the results of an analysis of the postoperative results in 500 patients, whom Miss Lucille Sobacke and he studied after surgery according to the amounts established by Dr. James W. White. The sudden death of Dr. Johnson that same year prevented presentation of the material. Through Miss Sobacke and Miss Jane Bailey, Dr. Cushman has used the same approach to study patients from her private practice as well as from the eye clinic at Northwestern University Medical School.

All patients had frequent examinations before and after surgery and the surgical results have been maintained. A few more than the charts show have obtained binocular single vision, for this was found at still later examinations. No squints secondary to previous surgery are included in this selection of cases.

For complete analysis of the muscle problem, frequent examination of any patient is necessary. This is especially true in the case of a young child whose squint is just developing and whose co-operation is not all that one might desire. It is well known that emotional stress resulting from apprehension or fear may increase the angle of a squint, especially one with a functional element, and so lead to false evaluation of the case. Repeated visits abolish all strangeness and the procedure becomes a game of following and blowing out a light—the examiner acquires the necessary information and the child his lollipop.

The intense desire of the parents to have immediate surgical correction of the squint may complicate matters. Needless to say, this never justifies surgery. It is much better to watch the squint develop and to make the correct diagnosis than to intervene prematurely and add to the difficulties already present. This is especially true in those cases complicated by an accommodative factor.

Another important condition which should be recognized and treated preoperatively is suppression amblyopia. It should be treated by complete occlusion of the sound eye and by stimulation and massage of the macula of the amblyopic eye until the fixation is steady and a satisfactory diagnosis can be made. During the early years, the use of atropine, glasses, and occlusion as indicated are used to develop alternate fixation.

Subsequent surgery should have a minimum of trauma as its goal. We do not advocate stripping the muscle of its capsule or completely severing its secondary attachments. Dissection is carried only to the point of exposure adequate for the surgical procedure.

Once the diagnosis is made and operation on the proper muscles is contemplated, the problem is—how much or how little surgery to do on the specific muscles. In the surgical treatment of squint, we have followed, as stated previously, a definite standard referred to as the *maximum and minimum amounts of recession or resection*. This standard is based primarily on the clinical experience of Dr. White, and on Dr. Johnson's and Miss

* From the Department of Ophthalmology, Northwestern University Medical School. Read before the Chicago Ophthalmological Society, May 18, 1953.

Sobacke's analysis, after surgical treatment, of 500 squint cases. Of secondary consideration are such anatomic factors of the extraocular muscles as insertion, check ligaments, and so forth.

We are aware of the feeling of those who state it is fool-hardy to decide prior to operation how much or how little surgery is to be done. In our opinion there is much less chance for error and confusion at the actual time of operation if a carefully preplanned program is followed. Such a program sets up a certain range of values which allows for any unusual findings at the time of surgery, as a mental picture has been made of the type of muscle which will be found.

MINIMUM AND MAXIMUM AMOUNTS

Recessions

Medial rectus—Central tenotomy to 3.0 mm.
Lateral rectus—4.0 to 7.0 mm.
Superior rectus—Central tenotomy to 4.5 mm.
Inferior oblique—Central tenotomy to 10.0 mm.+
Superior oblique—5.0 to 9.0 mm.
Inferior rectus—2.0 to 4.0 mm.

Resections

Medial rectus—3.0 to 6.5 mm.
Lateral rectus—6.0 to 7.0 mm. to 10.0 mm.+
Superior rectus—4.0 to 6.0 mm.
Inferior oblique—4.0 to 5.0 mm. and advance 8.0 to 10 mm.
Superior oblique—Tuck of 10 to 20 mm.
Inferior rectus—2.0 to 3.0 to 6.0 to 7.0 mm. and advance

These amounts are regulated according to the objective examination in eyes front and the diagnostic positions of gaze. The maximum amount is for those conditions which measure such large amounts as 35$^{\Delta}$ to 50$^{\Delta}$ in fields; as the measurements vary, a proportionate amount is done. The actual overaction or underaction must correspond to the measurements. Until all the findings can be correlated, surgery is not attempted.

These maximum and minimum values are offered only as a guide. In our hands they have worked well. It should be emphasized that no definite amount of resection or recession will correct a given number of prism diopters of squint. Too many factors, as the age of the patient, fixation habits, amblyopia, anisometropia, the accommodative factor, surgical technique of the surgeon, and so forth, are present for accurate prediction of a given result.

We do feel, however, that, in most instances, the maximum limits of these figures should not be exceeded—this is particularly true for recession of the medial rectus. In the majority of cases in which medial recession is contemplated, we favor the bilateral approach and feel that to exceed 3.0 mm. in young children is to lead to underaction of the medial rectus. A study of convergent squints followed over a period of 10 to 15 years after medial recession would be most interesting.

Using the objective method of examination and following the maximum and minimum values we have collected a series of 137 cases over a five-year period.

ANALYSIS

Total cases studied, 137

Binocular single vision perception, 93 (68 percent)
Postoperative eyes straight with alternation, 44 (32 percent)

In these cases the following center anomalies were present:

Convergence excess:	
partly or nonaccommodative	38
with secondary divergence insufficiency	44
Divergence insufficiency	17
Divergence excess	9
with secondary convergence insufficiency	12
Convergence insufficiency	9
Total	129
Remainder purely vertical	8

Involvement (paresis of one or more of the vertical muscles was present) as follows:

Superior rectus paresis	68
Inferior rectus paresis	19
Inferior oblique paresis	2
Superior oblique paresis	1

Tropias preoperative 102
31, Stable binocular single vision postoperative
27, Unstable binocular single vision postoperative

44, No binocular single vision postoperative

Phorias preoperative 35
- 31 Stable binocular single vision postoperative
- 4 Unstable binocular single vision postoperative

Binocular single vision postoperative 93
- 62 Stable
- 31 Unstable

No binocular single vision postoperative 44

31 Stable binocular single vision postoperative

- 8 Convergence excess—secondary divergence insufficiency
  - 1 Vertical anomaly
    - 1 Superior rectus paresis
- 13 Convergence excess accommodative partly nonaccommodative
  - 9 Vertical anomalies = Superior rectus paresis
- 5 Divergence insufficiency
  - 1 Vertical anomaly
    - 1 Superior rectus paresis
- 2 Convergence insufficiency
  - 2 Vertical anomalies
    - 2 Superior rectus paresis
- 3 Divergence excess—secondary convergence insufficiency
  - 2 Vertical anomalies
    - 2 Inferior rectus paresis

Total—31 center anomalies

- 15 Vertical anomalies
  - 13 Superior rectus paresis
  - 2 Inferior rectus paresis

27 Unstable binocular single vision postoperative

- 11 Convergence excess—secondary divergence insufficiency
  - 9 Vertical anomalies
    - 7 Superior rectus paresis
    - 2 Inferior rectus paresis
- 10 Convergence excess accommodative partly nonaccommodative
  - 3 Vertical anomalies
    - Superior rectus paresis
- 5 Divergence insufficiency
  - 4 Vertical anomalies
    - 2 Superior rectus paresis
    - 2 Inferior rectus paresis
- 1 Divergence excess—secondary convergence insufficiency
  - 1 Vertical anomaly
    - 1 Inferior rectus paresis

Total—27 center anomalies

- 17 Vertical anomalies
  - 12 Superior rectus paresis
  - 5 Inferior rectus paresis

44 No binocular single vision postoperative

- 25 Convergence excess—secondary divergence insufficiency
  - 16 Vertical anomalies
    - 13 Superior rectus paresis
    - 2 Inferior rectus paresis
    - 1 Superior oblique paresis
- 15 Convergence excess accommodative partly nonaccommodative
  - 8 Vertical anomalies
    - 6 Superior rectus paresis
    - 2 Inferior rectus paresis
- 1 Divergence insufficiency
  - 1 Vertical anomaly
    - 1 Superior rectus paresis with inferior oblique paresis
- 1 Divergence excess—secondary convergence insufficiency
  - 1 Vertical anomaly
    - 1 Inferior rectus paresis
- 2 Vertical anomalies only—no lateral imbalance
  - 2 Inferior rectus paresis

Total—42 center anomalies

- 26 Vertical anomalies
  - 20 Superior rectus paresis (one of these with inferior oblique paresis)
  - 5 Inferior rectus paresis
  - 1 Superior oblique paresis
- 2 Vertical anomalies only—inferior rectus paresis

PHORIAS PREOPERATIVE 35

31 Stable binocular single vision postoperative

- 4 Divergence insufficiency
  - 2 Vertical anomalies
    - 2 Superior rectus paresis
- 8 Divergence excess
  - 7 Vertical anomalies
    - 5 Superior rectus paresis
    - 2 Inferior rectus paresis
- 6 Divergence excess—secondary convergence insufficiency
  - 5 Vertical anomalies
    - 2 Superior rectus paresis
    - 3 Inferior rectus paresis
- 7 Convergence insufficiency
  - 5 Vertical anomalies
    - 5 Superior rectus paresis
- 6 Vertical anomalies only—no lateral imbalance
  - 6 Superior rectus paresis

Total—25 center anomalies

- 19 Vertical anomalies
  - 14 Superior rectus paresis
  - 5 Inferior rectus paresis
- 6 Vertical anomalies only—superior rectus paresis

4 Unstable binocular single vision postoperative

- 2 Divergence insufficiency
  - 2 Vertical anomalies
    - 2 Superior rectus paresis

1 Divergence excess
  1 Vertical anomaly
    1 Superior rectus paresis
1 Divergence excess—secondary convergence insufficiency

Total—4 center anomalies

3 Vertical anomalies
  3 Superior rectus paresis

AMBLYOPES 38 out of the 137

No binocular single vision postoperative 22
Unstable binocular single vision postoperative 8
Stable binocular single vision postoperative . 8
Vertical anomalies present in 34

22 No binocular single vision postoperative

12 Convergence excess—secondary divergence insufficiency
  6 Vertical anomalies
    3 Superior rectus paresis
    2 Inferior rectus paresis
    1 Superior oblique paresis
10 Convergence excess accommodative partly nonaccommodative
  5 Vertical anomalies
    5 Superior rectus paresis

8 Unstable binocular single vision postoperative

3 Convergence excess—secondary divergence insufficiency
  3 Vertical anomalies
    1 Superior rectus paresis
    2 Inferior rectus paresis
3 Convergence excess accommodative partly nonaccommodative
  No vertical anomalies
2 Divergence insufficiency
  1 Vertical anomaly
    1 Superior rectus paresis

8 Stable binocular single vision postoperative

2 Convergence excess—secondary divergence insufficiency
  1 Vertical anomaly
    1 Superior rectus paresis
5 Convergence excess accommodative partly nonaccommodative
  5 Vertical anomalies
    5 Superior rectus paresis
1 Divergence excess—secondary convergence insufficiency
  1 Vertical anomaly
    1 Superior rectus paresis

DISCUSSION

The total number of cases studied was divided according to the diagnosis, as the same type of surgery is carried out for each type, the only variation being in the amount.

The amount is decided upon according to the amount found in comparison to the maximum and minimum amounts already given, whether it be one half, one fourth, or one fifth of the maximum action in proportion with the action of the involved muscles.

For convergence excess in which the esotropia is always greater for near than for distance, with a close near-point of convergence, recession of one or both medials is indicated. If the esotropia measures 20^{Δ} or less the surgery is limited to one medial; if more than 20^{Δ}, both medials are recessed, and slightly more done on the medial which is more overactive as determined by measurements and appearance of the motility.

The diagnosis of convergence excess should be further modified by the terms accommodative and nonaccommodative or partially accommodative, which can be applied to most patients with hyperopic correction. If the medial rectus muscles measure and show as much overaction with as without the glasses and with a very close near-point of convergence (unlimited to 30 to 35 mm.), then it is nonaccommodative and the recession of the medial rectus muscles should be the maximum, otherwise a proportion of the maximum.

If the high plus correction reduced the esotropia for distance and especially for near a great deal and the near-point of convergence is not so close with the glasses as without them then the recession of one or both medials is reduced accordingly. The overacting medial or medials can be measured as well as seen.

With an associated accommodative factor, the surgery is determined after the full value of the corrective glasses has been found; therefore, the amount of recession of the medials is usually much less than in the nonaccommodative type.

If esotropia remains after the recession of both medials and the near-point of convergence comes out to 60 to 70 mm. after six months, any remaining esotropia is a divergence insufficiency and requires the resection of one or both lateral rectus muscles.

Divergence insufficiency in which there is more esophoria or esotropia for distance than near with a normal near-point of convergence (50 to 70 mm.) requires resection of one or both lateral rectus muscles. Here again if the esotropia measures more than 20$^{\Delta}$ for distance a binocular approach is made and both laterals resected, under 20$^{\Delta}$ one lateral is resected and in the proportions as outlined. No results seem to be obtained unless a resection of six to seven mm. is done, and 10 mm. is almost the maximum that can be done; therefore the surgery on the lateral must be regulated accordingly.

The resection of the laterals should not be done at the same step as the recession of the medials as time must be allowed for the laterals to pick up motility if their underaction is secondary to the overacting medial. It is frequently found to be the case that a suspected paretic lateral will become a normal acting muscle after the recession of the medial. Therefore the antagonists should not be operated upon at the same time but time allowed for the readjustment of the motility pattern.

Divergence excess, in which the exotropia or exophoria is greater for distance than near with a normal near-point of convergence, is always taken care of with recession of one or both lateral rectus muscles.

Binocular surgery is also used in divergence excess if the exotropia or exophoria measures more than 20$^{\Delta}$, and one lateral rectus only is recessed if the measurement is less than 20$^{\Delta}$. With borderline measurements judgement must be used, but more can always be done, and it is the best policy to do as little surgery as possible.

In secondary complications—such as convergence insufficiency secondary to a divergence excess, which comes on frequently in those patients with divergence excess associated with vertical anomalies—it is usually necessary to resect one medial rectus and recess one lateral (not of the same eye at the same time) according to the remote point of convergence and the motility of the laterals, recessing the more overactive lateral and resecting the more underactive medial.

Convergence insufficiency, as found in convergence excess secondary to wearing high plus lenses too long, should be treated by reducing or removing the plus in the lenses. Surgical correction may not be indicated in these patients.

Amblyopia must always be treated to improve the fixation of that eye before a satisfactory diagnosis can be made. Amblyopic treatment was carried out in 38 of the 137 patients and no satisfactory binocular vision was obtained in 22. Unstable binocular single vision remained in eight, and stable binocular single vision was obtained in eight to date. With eyes parallel the tendency is for the vision to continue to improve. A pure vertical anomaly should be taken care of as soon as the diagnosis can be made satisfactorily.

An associated vertical anomaly should be taken care of in the first step of surgery for a convergence or divergence anomaly, and this approach reduces the amount of surgery necessary as the lateral imbalance can vary a great deal in time, causing symptoms if the vertical is not taken care of early.

Our standard of binocular single vision postoperatively is for the corneal reflexes to be centered (parallel eyes), the muscle balance checked by the screen test in eyes front, measurement in fields and by use of the Worth four-dot test at 6.0 M. and 25 cm., and with comfortable function of the eyes.

The dissociation of convergence and accommodation cannot usually be consciously received until after seven years of age. Before that the glasses for the accommodative factor may need to be reduced to maintain normal vision and a normal muscle balance. A receding near-point or an increasing exophoria for distance and near are the indications to reduce the plus sphere in the glasses.

Phorias are treated the same as tropias as to the amount of surgery done for the muscle imbalance. The phorias do not require as much close observation following surgery as

the binocular single vision is easily maintained.

In the group of 137 patients, there were 38 with amblyopia in which the better eye was occluded during the treatment for one week to three weeks. The patch was worn continuously and changed only in our office or at the clinic. Macular stimulation was carried out during the office visit and the vision checked. When fixation became stable and vision had improved, a complete routine examination was possible and a diagnosis was made. The operative procedure followed as soon as possible, the child wearing the patch to the operating room.

After surgery both eyes were opened on the third day and the eyes checked for binocular vision. If there was a tendency for suppression the lens over the fixing eye was covered with clear lacquer as necessary, to stimulate the fixation of the amblyopic eye, and this was watched every three or four days, doing everything that was indicated to stimulate binocular vision.

Case reports

In closing, we should like to present a few case histories which exemplify some of the principles we have previously stressed.

Case 1

History. C. M., a boy, aged two and one-half years. Left convergence was noted at intervals, with fatigue for the past six months.

Refraction. R.E., +6.00; L.E., +6.00.

Ordered. R.E., +5.00; L.E., +5.00.

Screen test. cc E-ET 10 at 6 M.; E-ET 30 at 25 cm.

N.P.C. 30 mm. (left eye with glasses).

Fixation. Alternates easily at 6 M. and 25 cm. Binocular single vision part of the time.

Excursions. Slight overaction each medial rectus L > R. Suspect underaction right superior rectus.

After six months

Screen test. cc E 4 at M.; E 20 at 25 cm.

N.P.C. 25 mm. (left eye with glasses).

Fixation: Binocular single vision eyes front with glasses. Left convergence at near without glasses.

After one year

Glasses reduced to: R.E., +4.00; L.E., +4.00.

Screen test. cc No imbalance at 6 M.; E-ET 25 at 25 cm.

N.P.C. 45 to 50 mm. (left eye with glasses).

After fifteen months (after removing glasses for one month.)

Screen test: sc ET 45+; 6 M; ET 55, RH sl. 25 cm.

Diagnostic positions of gaze

Right	*Left*
ET 25	ET 35
LH 8-12	RH 6

N.P.C. 25 mm. (left eye without glasses).

After nineteen months

Screen test. cc E slight at 6 M.; ET 25, LH 8 at 25 cm.

Diagnostic positions of gaze.

Right	*Left*
ET 14	ET 20
LH 18	RH 4
ET 25+	ET 25+
LH 9	
ET 30	ET 30
RH varies	

NPC. 30 mm. (left eye with glasses: Right and Left, +2.00).

Screen comitance. Left medial rectus more overactive than right. Underaction each superior rectus —right predominant; secondary deviation left inferior oblique.

Diagnosis. Convergence excess accommodative partly nonaccommodative. Paresis right and left superior rectus.

Surgery. Recession left medial rectus 2.5 mm. Recession left inferior oblique 3.5 mm.

Comment. The case of C. M. was interesting because the squint appeared to be purely accommodative for the first year and he had single binocular vision with a good near-point of convergence with glasses. As the near-point of convergence began to recede the glasses were reduced to +4.00, then left convergence reappeared and increased and the left vision deteriorated. The diagnostic positions of gaze then indicated an underaction of the right superior rectus with secondary deviation of the left inferior oblique, as soon as this was definite and findings remained the same, a conservative recession of the left medial rectus was done with a 3.5 mm. recession of the left inferior oblique.

Since then the eyes have remained parallel without glasses by relaxing accommodation at near. He was advised to wear the glasses (right and left +2.00) for close work and as soon as he can appreciate the sense of relaxing accommodation the glasses will be removed entirely.

Case 2

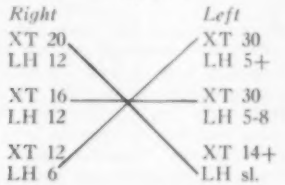
History. G. B., a boy, age 10 years, was seen several times during the years before the present history as his sister was a patient with an esotropia. His eyes were divergent for distance with no symptoms but when he began to have trouble at near and

the divergence for distance was almost constant the family wanted him treated.

Visual acuity. sc R.E., 20/20; L.E., 20/20—1. *Refraction.* R.E., +2.0D. sph. ⊃ +0.75D. cyl. ax. 120°; L.E., +1.75D. sph. ⊃ +1.0D. cyl. ax. 60° (no glasses ordered).

Muscle balance (without glasses). *Screen test.* sc XT 25+, LH 9 at 6M.; X-XT 12+, LH 6-8 at 25 cm.

Diagnostic positions of gaze



N.P.C. 100 mm. (left eye without glasses). Could be forced to 80 mm.

Screen comitance. Overaction each lateral rectus. Underaction right superior rectus; secondary deviation left inferior oblique. Slight underaction right inferior oblique.

Fixation. Prefers right eye (eyes front).

Diagnosis. Primary divergence excess. Paresis of the elevators of the right eye, right superior rectus, right inferior oblique.

Surgery. Recession right lateral rectus 5.5 mm. Recession left lateral rectus 5.5+ mm. Recession left inferior oblique 3.5 mm.

TWO YEARS POSTOPERATIVE

Screen test. Slight X *6 M.* and *25 cm.*
N.P.C. 70 mm. (left eye).
Excursions. No vertical imbalance in fields.
Prism convergence. 30Δ base-out at 25 cm.

Comment. According to our findings, the simple divergence excess and the vertical anomaly were taken care of at the same time with recession of the left inferior oblique. The underacting right inferior oblique has not increased and there was no vertical imbalance in the diagnostic positions of gaze two years after surgery and he has binocular single vision.

In young children, squints are frequently seen during their development. These cases should be followed until a definite diagnosis of the center anomaly and the associated vertical anomaly can be determined. When it is evident that the progression can probably be stopped, surgical correction may be undertaken. Uncomplicated divergence or convergence can often be treated nonsurgically for some time with atropine, occlusion, alternate patching, glasses, and so forth, postponing intervention until the child is old enough to co-operate in the diagnosis. When he is between four and six years of age, surgery for the correction of the vertical anomaly becomes a simple procedure. The following is a case in point:

CASE 3

M. P., a girl, aged three years, showed a divergence. No glasses were necessary. At the age of four years, the vertical anomaly became manifest and could be measured. At the same time the near-point of convergence was poor and she was developing a convergence insufficiency. Surgery was advised at this time but was postponed until she reached the age of eight years. By this time, she had diplopia at distance and near. Not until six months after surgery did she regain binocular single vision.

History. A divergence was noticed at intervals during the past year.

Screen test. sc X 30, RH varies at 6 M.; X 18 at 25 cm.

N.P.C. 60 mm. (right eye without glasses).

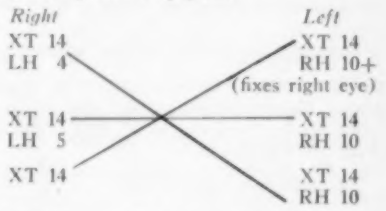
Excursions. No marked underactions or overactions.

FIVE YEARS LATER

Visual acuity. sc R.E., 20/25+1; L.E., 20/25+1. *Refraction:* R.E., +1.0D. sph. ⊃ +0.5D. cyl. ax. 90°; L.E., +1.0D. sph. ⊃ +0.5D. cyl. ax. 90°.

Muscle balance. Screen test. sc X — XT 18, RH 9 at 6M.; X 16, RH 3 at 25 cm.

Diagnostic positions of gaze.



N.P.C. 120 mm. (left eye without glasses).

Screen comitance. Underaction left superior rectus; secondary deviation right inferior oblique. Slight underaction right superior rectus. Left lateral rectus slightly more overactive than right.

Fixation. Prefers right eye 6 M. Binocular single vision 25 cm.

Diagnosis. Primary divergence excess. Secondary convergence insufficiency. Paresis right and left superior rectus.

Surgery. Recession left lateral rectus 5.5 mm. Recession right lateral rectus 4.5 mm. Recession right inferior oblique 3.0 mm.

SIX MONTHS LATER

Screen test. sc RH slight at 6 M. X slight at 25 cm.

N.P.C. 70 mm. (left eye without glasses). *Excursions.* No vertical imbalance in fields. *Prism convergence.* 15Δ to 20Δ base-out at 25 cm. *Worth four-dot.* Binocular single vision at 6 M. and 25 cm.

CASE 4

B. P., sister of M. P., aged eight years, had a divergence for distance since the age of four years, which was less noticeable after she started to school.

Her near-point of convergence could be stimulated with near-point exercises. With near-point exercises she could maintain a good near-point of convergence and, at the time of surgery, she could maintain binocular single vision for distance and near. After surgery, binocular single vision was present and has been maintained with adequate prism convergence.

History. Parents had occasionally noticed a left divergence but infrequently since she had been in school.

Visual acuity. sc R.E., 20/16—2; L.E., 20/15—2. *Refraction.* R.E., +1.5D. sph. ⊃ +0.5D. cyl. ax. 90°; L.E., +1.5D. sph. ⊃ +0.5D. cyl. ax. 90°.

Muscle balance. Screen test. sc X 20 (varies) LH 2-3 at 6 M.; X 12, LH sl. at 25 cm.

Diagnostic positions of gaze.

N.P.C. 105 mm. (right eye without glasses). *Screen comitance.* Right lateral rectus slightly more overactive than left. Suspect weakness right superior rectus.

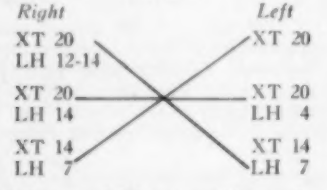
Fixation. Binocular single vision at 6 M. and 25 cm.

Treatment. Near-point exercises advised.

ONE YEAR LATER

Muscle balance (prolonged occlusion of left eye for nine days). *Screen test,* sc XT 20, LH 7 at 6 M.; XT 20, LH 4 at 25 cm.

Diagnostic positions of gaze.



N.P.C. 110 mm. (right eye without glasses). *Screen comitance.* Left lateral rectus overactive. Right medial rectus slightly weak. Underaction right superior rectus; secondary deviation left inferior oblique.

Fixation. Prefers left eye at 6 M. and 25 cm.

Diagnosis. Primary divergence excess. Secondary convergence insufficiency. Paresis right superior rectus.

Surgery. Recession right lateral rectus 5.0 mm. Recession left lateral rectus 5.5 mm. Recession left inferior oblique 3.5 mm.

TWO MONTHS POSTOPERATIVE

Screen test, sc X-XT 8 at 6 M.; X 4 at 25 cm.

N.P.C. 80 mm. (right eye without glasses).

Excursions. No vertical in fields.

Prism convergence. 2Δ base-out at 6 M. without glasses, 15Δ to 20Δ base-out at 25 cm.

CASE 5

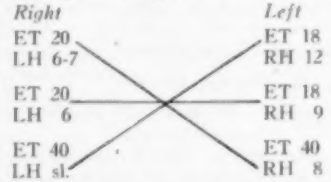
M. R., a boy, aged five years, had a satisfactory examination and diagnosis. At that time, the first step of surgery was carried out for the convergence excess (nonaccommodative) and vertical anomaly; the second step, for the secondary divergence insufficiency, was done six months later. Since then, he has maintained binocular single vision with normal vision although he has switched to a mixed astigmatism.

History. Right eye turned in since two years of age. Glasses and exercises did not help.

Visual acuity. sc R.E., 20/50—2; L.E., 20/33—1. *Refraction.* Wearing: R.E., +0.50D. sph. ⊃ +3.0D. cyl. ax. 105° = 20/13—3; L.E., +1.50D. sph. ⊃ +2.25D. cyl. ax. 75° = 20/20—1.

Muscle balance. Screen test. cc and sc ET 40, RH 5 at 6 M. and 25 cm.

Diagnostic positions of gaze.



N.P.C. 30 mm. (right eye with glasses).

Screen comitance. Right medial rectus more overactive than left. Underaction left superior rectus; secondary deviation right inferior oblique. Slight underaction right superior rectus.

Fixation. Prefers left eye at 6 M. and 25 cm.

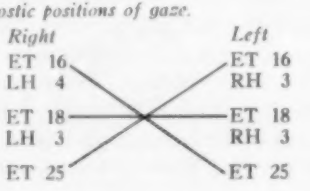
Diagnosis. Convergence excess mostly nonaccommodative. Paresis right and left superior rectus.

Surgery. First step: Recession right medial rectus 2.5 mm. Recession left medial rectus 2.0 mm. Recession right inferior oblique 3.5 mm.

SIX MONTHS POSTOPERATIVE

Muscle balance. Screen test. cc ET 15 at 6 M.; ET 20 at 25 cm.

Diagnostic positions of gaze.



N.P.C. 60 mm. (right eye with glasses).
Screen comitance. Right lateral rectus more underactive than left.
Diagnosis. Secondary divergence insufficiency.
Surgery. Second step: Resection right lateral rectus 10.0 mm.

FOUR YEARS POSTOPERATIVE

Refraction. R.E., $-1.50 : +2.50 \times 105^\circ = 20/20$ -2 L.E., $-1.25 : +1.75 \times 75^\circ = 20/20$.
Screen test. cc E 2-4 at 6 M. and 25 cm.
N.P.C. 60 mm. (right eye with glasses).
Prism divergence. 6$^\Delta$ base-in at 6 M. With glasses: 8$^\Delta$ base-in at 25 cm.
Comment. It must be emphasized that frequent refractions are necessary in all squint cases, since good vision is a real stimulation to binocular function.

COMMENTS AND DISCUSSION

Closing remarks by DR. CUSHMAN:* Sincere appreciation is offered in the memory of Dr. James W. White in this report. He died seven years ago in May, 1947.

Dr. White's great interest and meticulous study of the problems of the squint patient have been an inspiration to many students. He gave fully of his time to pass his knowledge on to his students.

The results in our hands of the approach herein reported have been satisfactory in 68 percent of the 137 cases, but I see no reason why the result should not be at least 80 percent, and we should strive toward this goal. As Dr. White said "there are some patients, it seems, who are not meant to have single binocular vision."

Making the measurements takes time. Sometimes, when handling a fist-full of prisms and with the variations that are found, one feels great frustration. The variations in themselves should not be depressing; the diagnosis usually remains the same. In convergence excess, the esotropia is always greater for near than distance, the near-point of convergence is close to very close, the overaction of the medial rectus muscles can be seen, and, usually, one muscle more overactive than the other can be noted.

The measurements in the six cardinal fields for any vertical imbalance are important. It is also important to have these measurements correspond with the motility of the muscles, as seen in the screen comitance maneuver.

In the patient with a phoria, prolonged occlusion of the more active eye may be necessary to bring out the real amount of the vertical anomaly.

Preference of the fixing eye accounts frequently for variations in the measurements in the fields of gaze.

Cosmetic surgery should be approached in the same manner as any other muscle problem. If possible, try to improve the vision in the poor eye by treatment of the amblyopia. This will aid in the diagnosis. Surgery should be regulated according to the near-point of convergence; usually recession of the medial rectus of the amblyopic eye and resection of the lateral rectus are necessary. When limitation of these muscles is obtained, it should cause no trouble.

The only exercises prescribed for these patients have been for treatment of amblyopia before attempting diagnosis of the muscle anomaly; this is to get better fixation of that eye for measurements. Some exercises for the poor near-point of convergence in convergence insufficiency were prescribed as part of the nonsurgical treatment. No preoperative or postoperative exercises were used.

Accommodative complications require full correction of the refractive error up to six months' time to obtain the best vision possible. By this time, it is usually possible to make a diagnosis of the muscle anomaly with the screen test and by measurement in the cardinal fields and of the near-point of convergence. That part of the squint not corrected by glasses is the nonaccommodative portion and requires surgery. Any vertical anomaly that is present at this time requires surgery.

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* My sincere thanks are expressed for the great help of Miss Lucille Sobacke and her students and Miss Helen Barth in reaching the goal we have reached so far, and to Dr. Willard for this present study.

When the near-point begins to recede, the plus correction must be reduced, and reduced as often as necessary to maintain a normal convergence-accommodation ratio.

The action of the underacting muscle, with the secondary deviation of the yoke muscle, should always show up in the screen comitance maneuver. Notation should be made of the fixing eye; this may vary in the fields and cause a variation in measurements.

The aim of the surgery is to make the eyes parallel; to improve vision; to change a tropia to a phoria with good vision in each eye and binocular single vision; to make a patient with an asthenopic phoria comfortable; and to improve appearance as well as vision.

Binocular approach and care of vertical anomalies seem to be successful. The evaluation of surgical results should not be considered as final for at least six months. We have had patients who have obtained binocular single vision with good vision in each eye as late as three to five years after this type of treatment.

25 East Washington Street (2).

PLASTIC CONSTRUCTION OF THE SUPERIOR PALPEBRAL FOLD*

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This paper aims to discuss the plastic construction of the superior palpebral fold in foldless eyelids. Incidentally, the operation can be made to widen the interpalpebral fissure while it somewhat corrects "slits" or "almond" eyes so common among the Orientals. The main objective of the procedure is to improve the physical appearance.

Nine of the 10 cases in this report were those of women, the 10th is that of a Chinese man who was born with mere slits for his eyes. Everyone teased him about his eyes with the comment that as he looked constantly sleepy, so his business too was just as sleepy. For this reason, he underwent the plastic operation and now that his eyes are wider he has lost that sleepy look. His business, too, has picked up.

In this operation, only the upper eyelid is considered. The main fold or wrinkle of skin in and around the upper eyelid is of great topographic interest. There is a horizontally curved palpebral or orbitopalpebral infold called the superior palpebral fold, dividing the lid into tarsal and orbital areas. The tarsal portion overlies the tarsal plate and the globe, while the orbital portion is related to the space between the tarsal region and the bony margin. The superior palpebral fold lies about two to three mm. above the highest point of the cutaneous insertion of the levator palpebrae superioris, the contraction of which muscle causes it to become deeply recessed, as the orbicularis oculi is thinnest along this line.

In this operation, one has to consider the following structures of the eyelid from without; the skin, the orbicularis oculi muscle, the septum orbitalis, the levator palpebrae superioris, and the tarsal plates.

PRELIMINARY EXAMINATION OF UPPER EYELIDS

1. Look for any ptosis, which is an absolute contraindication to the operation.

2. There should be no chronic infection of the eyelids, eyeball, tear glands, and nasolacrimal ducts and sac.

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* Presented at the annual meeting of the Philippine Ophthalmological and Otolaryngological Society, November, 1952, Manila, Philippines.

This paper was awarded the Medal of Merit for the best scientific paper presented at the annual meeting of the Philippine Society in November, 1952. Since that time up to November, 1953, a total of 40 patients have been operated on both eyes and two have been operated on one eye only.

Fig. 1 (Sayoc). Type of eye on which operation is performed.

Fig. 2 (Sayoc). Diagram showing incision which is made through the skin, subcutaneous tissue, and orbicularis down to the anterior surface of the tarsal plate.

Fig. 3 (Sayoc). Diagram showing excision of a narrow strip of the orbicularis (one to two mm.), extending from the nasal to the temporal ends of the incision.

Fig. 4 (Sayoc). Diagram showing suturing of the dermal layer of the lower skin flap to the anterior surface of the exposed tarsus.

Fig. 5 (Sayoc). Diagram showing the closed incision.

Fig. 6 (Sayoc). A sketch of an eye with a superior palpebral fold.

Fig. 7 (Sayoc). Results in typical cases. (Above) Before operation. (Below) After operation.

3. Look for the presence of blepharospasm.

4. Examine the texture of the skin of the lid. The presence of any loose hanging skin will modify the size of the fold.

Estimate of height and size of fold

This will depend on the aesthetic sense of the surgeon. For an oval face with big eyes, a bigger fold is suggested; while for a round face with tiny eyes, a smaller fold is more desirable.

The patient sits on a chair with a headrest, looking straight ahead. She closes her eyes and a line is drawn on the skin of the upper lid for the possible line of incision. With the arms of the small tissue forceps spread apart on the middle third of the lid along the drawn line of incision, slight pressure upon the forceps creates a temporary fold while the patient stares straight ahead upward. The other eyelid is identically treated.

Anesthesia

1. From the cutaneous side: After the instillation of two-percent pontocaine or five-percent coccaine into the conjunctival sac, the needle is thrust under the skin at the middle of the lid on the line, the point advancing gradually toward the nasal end. Without withdrawing the point of the needle at the center, it is then pushed gradually toward the temporal end.

2. From the conjunctival side: After instillation of two-percent pontocaine or five-percent cocaine to the conjunctival sac, the lid is everted and, with a tiny hypo needle, two-percent novocaine solution with a few drops of adrenaline (1:1,000) is injected under the loose conjunctiva just beyond the upper border of the tarsus until the middle of the eyelid is reached. Without withdrawing the needle, the lid is righted, and the needle point is pushed to the skin side. A sufficient amount of anesthetic is injected into the skin until both surfaces throughout the entire thickness of the eyelid are completely anesthetized.

Technique of operation

1. An eyeball protector covered with some ophthalmic ointment on the bulbar side is inserted under the upper lid with the assistant holding it in place. The modified chalazion forceps grasps the eyelid below the incision line. Holding the eyeball protector and the lid forceps with the left hand, an incision is made in one long-drawn stroke extending from the nasal to the temporal end, in case of the right eyelid, and vice versa, in case of the left.

The incision cuts through the skin, subcutaneous tissue, orbicularis, and down to the tarsus. A narrow strip of the orbicularis, from one to two mm., extending from the nasal to the temporal ends of the incision, is excised. This exposes the tarsus to which the skin has to be sutured.

2. With 6-0 chromic catgut or 6-0 braided black silk in an eye noncutting needle a stitch is taken in the dermal layer of the lower skin flap which is anchored on the anterior surface of the exposed tarsus, care being taken that the needle does not go through and through the thickness of the tarsus.

Three to five interrupted buried sutures are then made, encouraging adhesion of the dermis to the tarsus. To insure such adhesion, at times I touch the opposite dermal and the tarsal surfaces with trichloracetic acid, followed by alcohol, care being taken not to spill any.

The incision is then closed, using 4-0 black silk interrupted sutures. Strip dressings are applied on both lid incisions, narrow enough not to interfere with the opening and closing of the eyes. After operation, the patient can be up and about to attend to routine household duties.

Postoperative care

1. Since the operation is almost painless and the swelling of the upper lids is very slight, the patient does not have to stay in bed.

2. In case of some pain, a Demerol tablet may be given and an ice cap applied to the forehead.

3. The following day the wound is dressed; exposed to infrared rays for 30 minutes and for seven days daily; and the strip dressings are changed daily. Stitches are removed on the fifth to the seventh post-operative day. Healing is uneventful.

Comment

The same operation can be done in cases of blepharochalasis, the only additional step being excision of an elliptical piece of skin in the upper flap.

Summary

This is a preliminary report of plastic reconstruction of the superior palpebral fold in nine women and one man for aesthetic purposes. As far as I know, this is the first attempt locally to form a superior palpebral fold to enhance the physical appearance of those born with foldless eyelids.

Headquarters, 1st Station Hospital (PATC).

References

Alvis, W.: Modified Motais operation. Surgery of the Eye. Philadelphia, Saunders, 1940, p. 335.

Spaeth, E. B.: Surgical condition of the lids. Principles and Practice of Ophthalmic Surgery. Philadelphia, Lea, 1944, pp. 463-491.

Stallard, P. B.: The eyelids and reconstructive surgery. Eye Surgery. Baltimore, Williams & Wilkins, 1950, ed. 2, pp. 120-249.

Whitnall, E.: The structure and muscles of the eyelids. Anatomy of the Human Orbit. London, Oxford, 1932, ed. 2, pp. 129-162.

Ophthalmic Miniature

To Leopold Vohs, Book Editor, Hamburg

13 January 1885

Dear Sir:

I have not been able to get the proofs ready for the new edition of *Physiological Optics* but I have collected and arranged the literature which has accumulated in the meantime with the help of my assistant, Dr. Koenig. . . . However, there is so much literature that it might take up five galleys. This may not be as necessary as formerly in view of the now widespread circulation of the yearly reports of the *Medical Sciences*. . . .

Yours,

H. von Helmholtz.

From a letter in the collection of James W. Smith, M.D., New York, New York.

NOTES, CASES, INSTRUMENTS

USE OF SELSUN IN BLEPHARITIS MARGINALIS

LOUIS B. COHEN, M.D.
Philadelphia, Pennsylvania

Blepharitis marginalis, by description a subacute or chronic inflammation of the lid margins, is an extremely common disease. Since this area is the meeting place of the skin and the conjunctiva, the region tends to share in the affections of either, and the complexity of its follicular and glandular structures provides ready access and prolonged hospitality to any intruding organisms.

The purpose of this paper is not to discuss the many types of blepharitis marginalis but mainly blepharitis marginalis sicca and ulcerosa and the effect of Selsun ointment upon them.

It is an old axiom in medicine that, where there are many cures, usually none is particularly effective. This applies to the previous treatment of blepharitis marginalis, for which many medicines have been recommended but none with consistent success to various patients.

It is my feeling that with the judicious use of Selsun, ophthalmologists really can control blepharitis marginalis. Moreover, after one is convinced of its efficacy it will be surprising to realize the number of cases of blepharitis marginalis which have formerly been overlooked.

Selsun is the proprietary name of selenium disulfide. It is a chemical oddity in that its two constituents are located in the periodic table in the same way that the halogens are to each other. It has been used for some time, and in many cases, for seborrhea of the scalp. So far as could be found in the literature, there is no previous record of its being used in the treatment of blepharitis marginalis.

This study was inaugurated several years ago when I treated one of my patients for a stubborn case of blepharitis marginalis with the customary standard treatments and without much success. One day, bringing in her youngster for a refraction, she called my attention to her eyelids. It seems that in using some Selsun on her scalp, she had at times accidentally gotten some in her eyes. Following this, she noticed a marked improvement until now she was no longer bothered with the condition.

From this case, it seemed that Selsun would cure some cases of blepharitis marginalis without damage, but Selsun is too toxic to accept without verification. Rabbits were given instillations of 100, 50, and 25-percent ointments, using Borofax as a base, every four hours. The 100-percent solution damaged each rabbit cornea in which it was instilled within four days. The 50-percent solution damaged each rabbit cornea within a week but the 25-percent solution apparently produced no ill effects after one week's instillation.

At the same time that the rabbits were being treated, patients were given increasingly concentrated ointments, starting with five-percent ointment, to see if anything untoward happened. As nothing did, the final concentration decided upon was a 25-percent ointment heated to a melting point to obtain homogeneity. It was given every other night for four times, followed by a rest period of four to seven days. After this, if the condition persisted, the course was repeated.

The patients were told to remove all scales by vigorous rubbing with a moist wash cloth, and then to rub the salve into the lid margins only. They were told specifically not to put the salve into the lower cul-de-sac and then rub the excess over the lid margins as is so often done. The amount that can be absorbed

The animal experimentation work described in this article was performed by Robert Beisheim in the Research Laboratory of the Wills Eye Hospital.

is well within safe limits since the area of the lids times the contact time and the concentration is much smaller than for the scalp treatment.

No damage to the cornea or any idiosyncrasies have been observed. It should be noted that Selsun should be dispensed in a glass container as it reacts with metals.

Results

Statistically, 40 cases have been treated and followed up. They were divided into three classes: (1) Mild, consisting of scaliness only; (2) moderate, consisting of scaliness and redness; (3) severe, consisting of scaliness, redness, and loss of some lashes.

There were three cases of mild, 34 of moderate, and three of severe. The observation periods varied from three to 12 months. The results were divided into failures and cases that cleared, since the latter is the accepted term in dermatology. Of all the cases treated, all except three in the moderate group cleared. In some of the cases listed as cleared, there were recurrences, just as there are recurrences after use of Selsun on the scalp.

In the cases in which recurrences occurred the treatment was repeated and so far has been effective. Of the failures, two are unquestioned for, although the patients cooperated, there was no improvement, but the third, a nervous individual, stopped after one instillation because of burning in his eyes. There was no damage to cornea or lid margins in this case.

The percentage of cases cleared, 37 out of 40, or 92 percent is close to the result of Slepyan who cleared 87 percent his cases of seborrhea of the scalp with the use of Selsun in a much larger series of cases.

Case reports

There were three cases that stand out in this series and I should like to mention them briefly.

The first was that of an attractive young woman with a severe case of blepharitis marginalis. Because of its severity she was extremely self-conscious, wore dark glasses continuously, and her personality was becoming affected. After one course of treatment she was markedly improved and at the end of one month, resumed wearing uncolored lenses, had lost much of her self-consciousness, and was a very appreciative patient.

The second was that of a man who stated that he had been unable to obtain relief from a moderately severe case of two years' standing. According to him, the condition followed heavy doses of penicillin administered during a hospitalization. He responded to the use of Selsun and, months later, the condition was still cleared by repeated use of the Selsun ointment.

The third case was that of a boy, aged six years, who suffered recurrent styes and was unable to obtain relief. Following a course of Selsun, he did not have a recurrence in four months.

It would seem that enough data have been collected to show that Selsun used in the manner described is superior to that of any previous treatment of blepharitis marginalis and can safely be prescribed if the ointment is applied to the lid margins and with nothing to fear even if it should get into the eye.

An attempt was made to correlate an association between blepharitis marginalis and seborrhea of the scalp. However, there were too many cases in which, although scales were present in the scalp, the patients denied being bothered by dandruff. It was not possible, therefore, to set a standard to correlate seborrhea with blepharitis marginalis.

Similarly in taking scrapings, the results were unsatisfactory because most reports were negative.

Conclusions and Summary

1. A safe concentration of Selsun to apply to the lid margins in blepharitis marginalis has been determined.

2. An effective method for using Selsun has been described.

3. The results, 90 percent of the cases clearing, are better than any previously attained with other treatments.

4. An attempt to correlate blepharitis marginalis with seborrhea was unsuccessful.

1930 Chestnut Street (3).

REFERENCES

Abbott Laboratories: Brochure on Selsun.
Duke-Elder, W. S.: Textbook of Ophthalmology. St. Louis, Mosby, 1950, v. 5, p. 4950.
Slepyan, A. A.: Arch. Dermat. & Syph., **65**:228, 1952.

DIPLOPIA TECHNIQUE FOR OBTAINING BALANCED REFRACTION

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AND
WILLIAM H. HAVENER, M.D.
Columbus, Ohio

For the past eight years one of us (G. T. G.) has successfully employed the following technique to determine the proper balancing of spheric correction between the two eyes.

The subjective findings (by whatever method the examiner prefers) are left in place before the patient, and to this is added a plus 0.75D. sph., O.U. A four-diopter prism is placed base-up before one eye. The 20/40 line is now illuminated, and is seen vertically duplicated by the patient. If the refraction is accurate, both lines should seem approximately equally blurred.

If, as illustrated in Figure 1, one line is less blurred than the other, an additional plus 0.25D. sph. is added before the eye seeing the more clear image. This will generally suffice to produce equal blurring; if not, another plus 0.25D. sph. is added before the more clear eye.

If more than plus 0.5D. sph. must be added before the clearer eye, the refraction is in considerable error, or the worse eye has noncorrectibly reduced vision. At the point of equal blurring, removal of the plus 0.75D. sph., O.U., should make both lines equally clear. Remove the prism. Whether the patient will accept an additional plus 0.25D. sph. binocularly may be checked, especially if maximum plus correction is desired as in a prepresbyopic or an esophoric patient. The trial frame should now contain the final prescription.

Suppression of one eye is made evident by failure of the patient to perceive the two images. Rarely a vertical muscle imbalance will neutralize the prism displacement, permitting fusion. Reversing the prism produces marked image separation in such a case. Monocular decreased vision, uncorrectible with lenses, is the only contraindication to use of this diplopia technique.

This is essentially a "fogging" method, and is especially helpful in a noncycloplegic refraction. When one eye is covered, many patients are prone to accommodate, often a different amount for each eye. Although it is accepted that accommodation must be equal in the two eyes, under monocular conditions there may be more accommodation when the more hyperopic eye is uncovered than when the less hyperopic eye is uncovered. This difference in the amount of accommodation cannot happen under binocular testing conditions, and a truly balanced refraction is obtained.

CASE REPORT

The following case is an example of the application of the diplopia technique.

W. B. (hospital number 759793 U.M.), a 25-year-old white man, was refracted under paredrine (one percent), homatropine (five percent) cycloplegia as:

1. O.D., $+2.0$D. sph $\bigcirc$ $+1.25$D. cyl. ax. $85^\circ = 20/15$; O.S., $+5.0$D. sph. $\bigcirc$ $+0.25$D. cyl. ax. $180^\circ = 20/15$.

At postcycloplegic examination, done by examining each eye separately, the patient accommodated more with the left eye, giving:

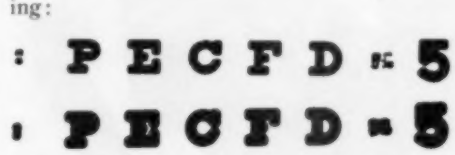

Fig. 1 (Gwathmey and Havener). Unequally blurred images, indicating spheric imbalance between the corrections of the two eyes.

2. O.D., +0.5D. sph. ⊂ +1.25D. cyl. ax. 85° = 20/15, J0 at 15 cm.; O.S., +1.5D. sph. ⊂ +0.25D. cyl. ax. 175° = 20/15, J0 at 30 cm.

Use of the diplopia technique produced the following balanced prescription with a latent hyperopia of 1.5D., O.U.

3. O.D., +0.5D. sph. ⊂ +1.25D. cyl. ax. 85° = 20/15, J0 at 15 cm.; O.S., +3.5D. sph. ⊂ +0.25D. cyl. ax. 175° = 20/15, J0 at 15 cm.

Upon removal of the prisms, the patient experienced immediate clear binocular single vision, without aniseikonic symptoms. The addition of plus 0.25D. sph., O.U., was not accepted. This prescription (3) was given, and has been worn comfortably since. If manifest refraction alone has been done, the left eye would have been relatively undercorrected to a considerable degree. A careful examiner would have been alerted by the remote near point O.S.; nevertheless, the patient would not accept more sphere O.S. monocularly.

Apparently in anisometropia there exists a conditioned reflex to accommodate to a greater degree when the more hyperopic eye alone is uncovered. (Throughout life, covering of the more emmetropic eye has necessitated additional accommodation for clear vision with the more hyperopic eye.)

Clinically we have consistently found a larger latent hyperopia in the more hyperopic of the two eyes in cases of anisometropia being tested monocularly.

SUMMARY

A prism-diplopia technique is described for comparison of the images seen simultaneously by the two eyes. Since both eyes must be accommodated to the same degree at any given time, this method insures a properly balanced refraction.

417 East Robinson Avenue.
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PORTABLE TRANSFORMERS FOR DIAGNOSTIC INSTRUMENTS*

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There has been a need for a small, lightweight transformer for use with electric ophthalmoscopes and similar instruments. This would be particularly valuable on hospital wards, as well as in clinics or at patients' homes.

A new model portable transformer unit, mounted within a small rectangular box, was developed at this laboratory for use at the Medical Center, supplying any or all of the following voltages (alternating current): two to three for most American-made ophthalmoscopes and retinoscopes; four to six for gonioscopes, the American Optical Giantscope and so forth; and eight to twelve for the Keeler Pantoscope, and the like.

Each unit contains a voltmeter to indicate the voltage being supplied to the attached instrument, and a rheostat for brightness regulation. An on-off switch was incorporated with the rheostat, and a small pilot-light included to remind the user when the current has been left on.

The entire unit can be held comfortably in one hand, and may be carried easily in the physician's bag or pocket. The cord, to be

* From the Ophthalmology Research Laboratory (Dr. I. H. Leopold, Director), Albert Einstein Medical Center, Northern Division. This work was made possible by a grant from the Weinstock Fund. These portable transformer units are being manufactured by Medico Instruments, 1220 North Broad Street, Philadelphia, Pennsylvania.

Fig. 1 (Askovitz). A portable transformer.

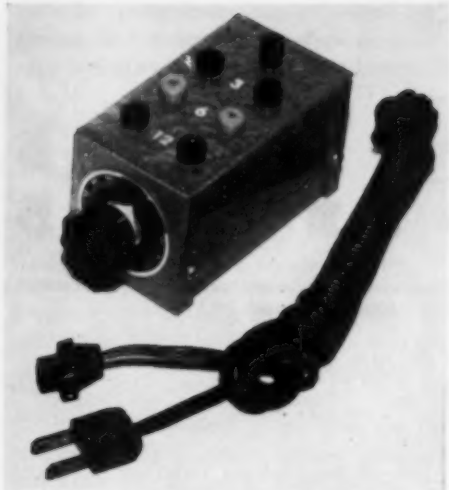

Fig. 2 (Askovitz). Another model of portable transformer.

plugged into a wall outlet, is detachable from the box for convenience of transportation. Somewhat smaller models have been made without the voltmeter. Representative units are shown in Figures 1 and 2.

Albert Einstein Medical Center (41).

INTRACAPSULAR FORCEPS*

R. Townley Paton, M.D
New York

The intracapsular forceps shown in Figure 1 were designed to facilitate grasping the capsule at either the upper or lower pole. Because of the angulation and curvature of the forceps it is easier to grasp the lens at either the upper or lower pole. Once the capsule has been grasped, less rocking or side-to-side motion is necessary to break the zonular fibers.

The cupped jaws of the forceps are somewhat smaller than those in the ordinary Arruga forceps, and there is

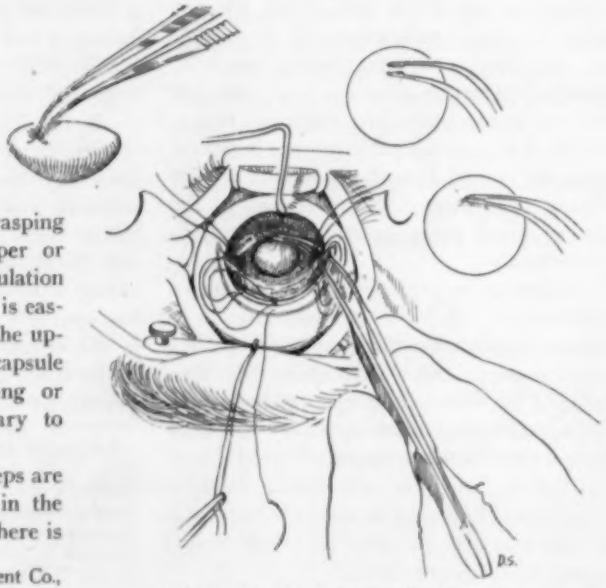

Fig. 1 (Paton). Intracapsular forceps.

* Manufactured by Storz Instrument Co., Saint Louis, Missouri.

a stop which prevents the forceps from opening more than a few millimeters. This stop idea is of course not original, as it has been used on many other types of forceps. Because of a slight shearing effect that is obtained when the jaws are brought together, many capsules may be grasped with this forceps when other forceps fail to do so.

927 Park Avenue (28).

Ophthalmic Miniature

Brisseau says that this disorder (glaucoma) is an opacity of the vitreous-humour joined to a cataract; so that in this case, although you depress the crystalin, yet the opaque vitreous-tunick still hinders a passage to the rays of light. To illustrate which, you have the history of M. Bourdelote, the King's Physician, who had for some years laboured under a perfect cataract in one of his eyes. At his death he ordered that the diseased eye should be examined, in order to clear up the disputes about the true seat of a cataract. This was done by Mareschal, the King's first Surgeon; who found the crystalin-lens opaque and solid with its nucleus of centre of a more solid consistence and yellower colour than its circumference. The sinus, or cavity, of the vitreous-humour, in which this body was lodged, was clouded almost a line in depth; and this he concludes to be a glaucoma; because says he, though the cataract were depressed yet the opacity of the vitreous-membrane would still stop up the passage of the light. Therefore this agrees with the observations of most authors, in its being an incurable disorder.

Silvester O'Halloran of Limerick,
A New Treatise on the Glaucoma, or Cataract,
Printed by S. Powell, Dublin, 1749.

SOCIETY PROCEEDINGS

Edited by DONALD J. LYLE, M.D.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

June 8, 1953

DR. J. W. MCKINNEY, *presiding*

IRITIS IN SARCOIDOSIS

DR. OSCAR DAHLENE presented a 69-year-old Negress who was first seen in the John Gaston-Gailor Clinic in January, 1952, complaining that her eyes were red and sore, and her vision blurred. Vision was 20/50, O.U. Treatment with zinc-sulfate drops alleviated the condition. She was seen again in July, 1952, with the same complaint of burning and irritated eyes and was again treated with zinc-sulfate drops.

On October 1, 1952, she returned with new complaints. The symptoms were headache, aching of the left eye, episodes of redness, and severe aching of the left eye, lasting all night, with blurring of vision, and gradual clearing of the vision as the pain and redness receded. The eyes were white at that time.

Tension was: R.E., 13 mm. Hg; L.E., 25 mm. Hg (Schiøtz). A water-drinking test was performed, and the maximum tension recorded was R.E., 13 mm. Hg; L.E., 32 mm. Hg. The patient was placed on pilocarpine (two percent) in the left eye, and the tension seemed to respond.

On October 31, 1952, she returned with an acute attack of iritis, showing greasy keratic precipitates, thick flare, lardaceous yellowish-white nodules of the iris and an oval, fixed pupil. Vision was: O.D., 20/50; O.S., 20/200; tension: R.E., 13 mm. Hg.; L.E., 44 mm. Hg. This time she was placed on atropine and hot compresses, and in three days showed much improvement, with tension falling to R.E., 13 mm. Hg; L.E., 20 mm. Hg.

The usual rundown of possible causes of iritis was begun. Serologic test for syphilis was negative; chest X-ray films showed some calcification of the aorta and minimal fibrosis of the hilar region; X-ray films of the hands and wrists showed slight arthritic changes essentially normal for her age, and skull films were normal. A Toxoplasma test was reported positive in a dilution of 1:256, which is not significant in an elderly person of this area. The Frei test was negative, as was the test for brucellosis, agglutinations for typhoid, proteus OX-19, tularemia, and undulant fever. No lesions were seen in the fundus of either eye.

On January 30, 1953, she was admitted for further studies. Electrocardiograms and physical examination revealed a mild cardiac decompensation which was treated appropriately. Lumbar puncture was done, and the spinal fluid studies were normal. Serum calcium, alkaline phosphatase and inorganic phosphorus, serum chlorides, and bromsulphalein tests were also within normal limits. Cephalin flocculation was three plus but prothrombin time was essentially normal. The liver was punched twice to obtain tissue for biopsy, and this showed only normal tissue. A lymph node was also found to be normal. Skin from some old degenerated and pigmented areas on the legs showed chronic granulomatous inflammation of the skin with giant-cell formation, consistent with sarcoidosis.

On February 12, 1953, it was noted that there were synechias of the right iris and, upon examination with the biomicroscope, Koeppe nodules and pigmented keratic precipitates were seen on the margin involved in these synechias. She was given local cortisone drops (1:4) and atropine for both eyes. The redness of the eyes subsided rapidly. Tension was: R.E., 20 mm. Hg; L.E., 17 mm. Hg.

The case was discussed at the medical seminar, and the consensus was that sarcoidosis was the cause of the iritis. Systemic therapy with cortisone was advised and, on February 17th, a regime of 100 mg. daily intramuscularly was established. By February 19th the keratic precipitates were crenating and the ciliary injection was almost gone. On February 23rd and 24th, the nodules of the iris were small and now had vanished except for a small white streak on the nasal iris of the left eye. The dense posterior synechias remained. Tension was normal.

In summary, the findings supporting the diagnosis of sarcoidosis are the eye picture, anemia, low white blood count with elevation of basophils and eosinophils, A/G ratio reversal and hyperglobulinemia, negative PPD, and the skin biopsy.

Unusual case of esotropia

Dr. Philip Meriwether Lewis presented a patient recently operated upon for an "acute" concomitant strabismus.

B. J. L., a white girl, aged six years, was first seen in June, 1949, because of recurrent hordeoli. There was no history of her eyes crossing nor was any noticed by this examiner. The child was not seen again until March, 1952, with the following history:

Four weeks previously she had been ill with chickenpox and influenza. During this illness her mother noticed that her right eye was crossing. Thinking that the crossing was a temporary thing from which she would soon recover she waited for four weeks before bringing her in for examination.

Examination showed vision of 20/20, O.U. There was a 25 prism diopter esotropia for distance and 20 for near. She fixated with the left eye. There was no apparent limitation of motion and measurements in the cardinal fields showed that the angle of deviation remained the same in all directions of gaze. There was no vertical deviation. There was true fusion and stereopsis on the synoptophore at 25 prism diopters base-out. With a red glass, the separation of images remained the same in all directions. If there had been a paresis of the right lateral rectus, it had apparently cleared up completely. The child was conscious of diplopia most of the time but it did not annoy her. Refraction under atropine showed one diopter of hyperopia without astigmatism, in each eye.

A full correction (+1.0D. sph., O.U.) was prescribed and worn constantly for about eight weeks. There was no change whatever in the angle of deviation. There was no longer an awareness of diplopia, but it could be brought out easily with a red glass. Feeling that no improvement would occur without operation, a resection of seven mm. of the lateral rectus and a recession of four mm. of the medial rectus of the right eye were performed.

At first the eyes were straight for near, but there was an esotropia of five prism diopters for distance. She suppressed the image of the right eye most of the time but apparently used them together for near. The orthoptist and I both feared that a continuation of orthoptic training might develop a disturbing diplopia for distance so it was completely abandoned.

One month later, seven weeks after operation, her ability to use her eyes together had spontaneously returned for distance as well as near. She was allowed to leave her glasses off most of the time. Eight weeks after that, about four months after operation, her muscle balance was normal and she had good fusion and stereopsis. Glasses were abandoned entirely.

In my experience this case is unusual. A concomitant strabismus developed suddenly and remained the same until corrected by surgery. Orthoptics did not seem likely to be helpful and were discontinued. Complete recovery then occurred spontaneously.

Diagnosis sought

Dr. M. I. Shathir presented a seven-year-old white girl who had been brought

to the eye clinic because of blindness of the right eye. About 10 months ago, the patient's uncle noticed a peculiar whitish reflex in the pupil of the right eye. The mother patched the left eye and found that the right eye was absolutely blind.

The patient was well developed at birth and weighed seven pounds. She had an attack of measles at the age of six weeks. No history of influenza, whooping cough, ear disease, tonsillitis, fits, unconsciousness, or attacks of screaming was given.

Eye examination revealed a normal left eye. The cornea of the right eye was bright; the anterior chamber shallow; the pupil irregular, slightly dilated, inactive to direct and indirect light with presence of many posterior synechias. The lens was clear. There was a whitish mass occupying the whole vitreous, very close to the posterior surface of the lens. The fundus could not be visualized. Tactile tension was soft, and the vision in this eye was nil. Transillumination under general anesthesia revealed no area of increased density except for a possible area at about the 5-o'clock position.

Laboratory tests: Blood was negative except for a slight leukocytosis, 17,900, and an increase of esinophils, seven percent. Urine and stool were negative. Kahn and PPD were negative.

The patient was hospitalized and on May 11, 1951, an incision was made at the 5-o'clock position about five mm. behind the limbus through the sclera with a cataract knife and a needle was inserted into the vitreous and about 0.6 cc, of a cloudy yellowish fluid was obtained. The fluid revealed a small number of leukocytes and numerous eosinophils. Culture was negative.

The purpose of presenting this case is to seek a possible diagnosis and treatment.

Gradual visual loss

Dr. J. M. Davis reported the case of Mr. G. D. L., aged 45 years, white, with gradual loss of vision of the left eye. The patient entered the clinic on October 24, 1952, with the complaint of gradual loss of vision, O.S., over a period of eight or nine months. His general health had been good. There had been no eye infections nor injury. He did sustain a blow from a baseball three years ago.

Vision was: O.D., 20/25, J14; O.S., light perception only. Corneal sensitivity equal; pupils equal and each reacts promptly to direct and consensual light and to accommodation; extraocular muscles, normal; tension, 18 mm. Hg, O.U.; fundi normal; refraction, O.D., +2.5D. sph. ⊂ +2.5D. cyl. ax. 180° = 20/25+; add, +1.5D., J1. Visual fields of the right eye were normal; several malingering tests were normal (correct response). X-ray films of the orbit, orbital apex, and skull were normal. Kahn, negative; routine blood and urine tests, normal. A consulting neurologist found no neurologic difficulties. After treatment with vasodilators and multivitamins for one month, there was no improvement.

Daniel F. Fisher,
Recorder.

YALE UNIVERSITY CLINICAL CONFERENCE

May 8, 1953

Dr. R. M. Fasanella, *presiding*

General surgeon and eye lesions

Dr. William H. Ryder, New Haven, Connecticut, presented four cases of eye lesions in which general surgery was necessary to clear the eye condition.

The causes of eye diseases are manifold and a good part of the time the cause is never determined. Among the reasons for this is the lack of thorough investigation. In those cases which are thoroughly investigated and the cause determined, it may become necessary to call in the general surgeon.

This paper will bring out four cases, as examples of the need of surgery, other than ocular surgery. This group will consist of two cases of tuberculosis of the eye men-

tioned primarily because it is believed that ocular tuberculosis occurs rarely, with an active lesion elsewhere in the body. This might be more understandable by quoting the following from Duke-Elder: "It may be taken, therefore, that with the exception of acute miliary tuberculosis, ocular tuberculosis occurs with the greatest rarity in the presence of active tuberculosis elsewhere, but rather in apparently normal, healthy, well-nourished individuals with a healed or benign tuberculous infection in some organ, usually the lymphatic system in the chest, more rarely in the lungs, the glandular depot, even although it is usually slight, quiescent, clinically healed or calcified, being capable of disseminating infection after the primary pulmonary focus has healed, presumably by the occurrence of an intermittent bacilloemia which is usually without systemic significance."

The diagnosis of tuberculous eyes may often be confused with Eales's disease, which is a recurrent intraocular hemorrhagic condition of unknown cause that appears in the eye. This disease is seldom limited to one eye.

Due to the fact that rigid investigation was required in the cases to be reported, the method of each investigation will be gone over briefly and, in doing so, the need of general surgery will be brought out.

Case 1. A white man, aged 37 years, was seen April 4, 1936, complaining of blurred vision in the left eye. History revealed that, in November, 1928, he had a pulmonary hemorrhage following which he was admitted to a sanatorium on December 26, 1928, and discharged May 31, 1929, with the discharge note of moderately advanced pulmonary tuberculosis, satisfactory convalescence, condition quiescent. Follow-up treatment was observed until December, 1934, when he was again admitted to the sanatorium. Examination revealed a definite extension of the lesion on the right side.

On January 25, 1935, a phrenicectomy was done on the right side. On January 30, 1935, he was again discharged somewhat improved, with positive sputum and an open cavity on the right side. Two weeks before discharge, he complained of a white blur in front of his left eye. He was told by an oculist that this was a retinal hemorrhage. On treatment, his eye cleared in three weeks only to leave floating cobwebs before it. These cobwebs progressively became worse until they coalesced to occlude the vision totally. On April 4, 1936, he was advised to again seek sanatorium treatment and was admitted to the William Wirt Winchester Hospital, April 13, 1936, where the following treatment took place:

Tuberculin therapy from April, 1936, to October, 1936, with apparently some improvement. On October 25th, the right eye began to show extensive changes in the periphery of the choroid but was free of exudate and the vitreous was clear.

Prolonged bedrest was continued until February 8, 1937, when he was transferred to the New Haven Hospital where thoracoplasty consisted of a first stage on the right, at which time all posterior portions of the first, second, and third ribs and a large part of the anterior portion were removed. The patient did not stand the operation well. During the postoperative course, the temperature rose to 102°F. to 103°F.; there was paroxysmal dyspnea, with slight cyanosis, suggesting intermittent bronchial obstruction. On March 4th, the second stage was done with a resection of the fourth and fifth ribs.

The patient was discharged March 18, 1937, from the New Haven Hospital and was readmitted to the Winchester Hospital the same day. Improvement in all his conditions was noted following this readmittance to Winchester Hospital, from which he was discharged December 15, 1938.

He again consulted me on January 31, 1939. Both eyes were quiescent and the vision was: R.E., 20/20−1; L.E., 20/50; both eyes together, 20/15. There was an error of vision in the left eye, corrected by 0.25 diopter, far-sighted or hyperopic astig-

matism giving 20/40 as vision, L.E. Vision in right eye was 20/20 with no error. The fundi showed several quiescent areas of chorioretinitis.

The result of all this treatment has rehabilitated this man so that he can carry on a regular position; he is again living with his wife and son, the latter being only nine months old when he went to the sanatorium. Today, he shows no active areas of tuberculous involvement.

Case 2. A white woman, aged 34 years, a trained nurse, was first seen May 21, 1934, with the complaint of blurred vision with black floating spots in the right eye. Examination revealed the media, hazy and blurred, with floating opacities. The disc was negative and the upper nasal field showed a large area of active choroiditis. Local treatment was instituted and the patient referred to the family physician for a physical check-up and to a clinical laboratory for blood chemistry and urine tests, including Mantoux test and X-ray studies of her sinuses, teeth, and chest. The reports of all this work yielded a positive Mantoux test and pus cells in the urine.

Tuberculin therapy was instituted and this, with the local treatment, apparently prevented the spread of the lesion. The next treatment was foreign-protein therapy which did not apparently improve the condition, so a cystoscopy was resorted to on October 8, 1934.

Treatment was continued with tuberculin and the condition still persisted but was not too active. At times, synechia or adhesions would form, which could be easily broken up.

Treatment continued until July 11, 1935, when a catheterized specimen of urine was sent to the laboratory for inoculation into a guinea pig. The guinea pig died August 30, 1935. An autopsy revealed numerous acid-fast organisms of the inguinal gland.

The patient was again admitted to the hospital September 13, 1935, for a second cystoscopy. Specimens were taken at this time from the bladder, the right ureter, and the left ureter, and each specimen was injected into individual guinea pigs.

The guinea pig inoculated with the bladder specimen was autopsied and was positive for acid-fast organisms in the spleen and the lymph nodes. The guinea pig with the right ureter inoculation was negative for any pathologic process. On November 22, 1935, the left-ureter inoculated guinea pig showed acid-fast organisms in the spleen and lymph nodes.

The patient was immediately referred for advice on removing the left kidney and, on April 21, 1936, a nephrectomy was done. After the operation, gross examination showed an externally normal appearing kidney but, on cut section, a small area of tuberculous necrosis was found at the upper pole. Following the removal of the kidney, the active infection in the eye quieted down but, as yet, the vitreous opacity persists.

This patient developed a sensitivity to atropine. In 1940, she had a slight flare up of the choroiditis which quieted down when two abscessed teeth were removed. This occurrence shows that these cases should be kept under constant observation, as any associated infection might possibly stir up the old tuberculous condition.

I personally feel that local infection plays a great part in diseases of the eye and, in all probability, the search for many of the foci is discontinued before any one of them is found. Chief among the foci are the tonsils and the teeth.

Case 3. A white man, age 31 years, a physician, was seen July 27, 1939. He complained that the right eye had been red and inflamed for three weeks with slight pain, no discharge, and blurred vision. Vision was: R.E., 20/50+2; L.E., 20/15−1. Examination revealed an acute iritis. After several atropine treatments, the synechia broke up, the pupil became fully dilated and the inflammation subsided. X-ray films of the teeth revealed an abscessed tooth, which was removed. There was no further trouble

with the eye until September, 1939, when the right eye again became inflamed.

Hospitalization was recommended and a complete physical, otolaryngologic, blood chemistry, X-ray and Mantoux tests were done, giving only a diagnosis of deviated nasal septum. A submucus resection was done September 25, 1939. On September 27th, the patient had a chill and elevated temperature and felt grippy. By October 4th, his general condition was better and he was discharged on October 9th, with the eye white, pupil dilated, and media hazy.

On October 18, 1939, he entered the Manhattan Eye, Ear, Nose, and Throat Hospital, where he was believed to have an ethmoid sinus condition, and the ethmoids were operated upon. This did not relieve the active condition in the eye and since a secondary glaucoma had developed, due to the synechia or adhesions forming, an iridectomy was done. He was discharged from the Manhattan Hospital, with the eye still active and cataract formation already well advanced. About two weeks after this, he developed an ischiorectal abscess and, following incision and drainage of this condition, the infection in this eye quieted down, and to date he has had no trouble with his eyes.

After this operation had been done, he recalled that while interning in 1935, he had had a small ischiorectal abscess, which quieted down without incision and drainage. This fact, not in his history, should have been noted to aid in the diagnosis. A careful history is absolutely essential and should be emphasized to a greater extent.

Case 4. G. K., a white woman, aged 21 years, a machine operator, was seen on March 10, 1941. Vision was: R.E., 10/200; L.E., 20/30−2. For the past two weeks, the right eye has been blurred.

Examination revealed cloudy media so that the fundus could not be examined. Slit-lamp examination revealed a hazy cornea due to deposits on Descemet's membrane, good anterior chambers, no increase in tension. The diagnostic impression was a uveitis.

The teeth were X rayed and it was found that the upper right first central incisor, the lower right six-year molar, and the upper left third molar, were abscessed. These were removed. The only other signs were moderately large tonsils, appearing not too active, and a positive Mantoux test.

Sulfanilamide was started but had to be stopped, due to blood changes. She was given two injections of typhoid vaccine with good reaction. She was discharged improved, March 25, 1941.

The vitreous in this case did not clear, although the active signs had quieted down. Tuberculin therapy was instituted starting with 1/100,000 dilution. This did not aid the condition and on May 27, 1941, the drainage increased and a paracentesis was done. This seemed to relieve her condition and on July 19, 1941, a tonsillectomy was done following which, all activity and irritation stopped. The eye now is smaller in size, has a mature cataract, annular synechia or adhesions of the pupil, good light perception and projection, and normal tension.

Summary. The cases reported showed tuberculosis and focal infection which needed some form of general surgery to relieve them. My wish is to bring these cases before you as examples of the extremes that must be gone to in investigation and treatment of eye diseases, and also the value of some type of general surgery to relieve the disease.

William J. Glass,
Recording Secretary.

MADRID OPHTHALMOLOGICAL SOCIETY

April 23, 1953

Dacryostomy

Dr. Sylvan, of Pamplona, spoke briefly on "Dacryostomy in the absence of the lacrimal sac," describing a new technique,

which is an improvement on the one he showed at the 30th Congress of the Spanish-American Ophthalmological Society in Cadiz, September, 1952. He showed a sketch of a catheter which will not be clogged up by exudates.

Tapetoretinal degeneration

Dr. Suner, of Madrid, spoke on tapetoretinal degeneration in sisters, aged 15 and 11 years, who showed these lesions in the fundus. He described the clinical histories. The ophthalmoscopic picture in the older sister showed atypical pigmentary degeneration of the retina in both eyes, atrophy of both optic papillas, and bilateral macular degeneration. In the younger, degeneration of both maculas without pigmentation was seen. He emphasized the absence of consanguinity between the parents and grandparents and said that both patients had had good vision and had learned to read in their early years. The older sister was enrolled in a school for the blind, and the younger one had an acuity of 1/40.

To justify inclusion of the two cases in a single pathologic entity the speaker mentioned the anatomic-pathologic lesions of these degenerations as described by various authorities. He reviewed the theoretical and etiopathogenic principles, dwelling largely on the factors of heredity, endocrine disturbances, and the abiotrophy of Gowers and his followers.

He concluded by stressing the necessity of differentiating from the congenital malformations this other type of heredodegenerative lesions which appear in fully developed tissues. He ended with the thought that perhaps there was an abnormality in the biotrophy of the retinal neuro-epithelium in these patients.

Discussion. Dr. Arjona said that these cases of macular degeneration demonstrate the close relation which exists between pure macular degenerations and the group of degenerations which Leber included in the group of tapetoretinal degenerations in which pigmentary degeneration is the most prominent symptom.

We see that many times the degenerative process does not remain limited to a system which is embryologically related to the retina, tions of other organs which may accompany as is the nervous system. There are affecthese processes in the retina, in some 10 percent of cases, such as deafness through degeneration of the organ of Corti, imbecility through degeneration of the ganglion cells in the cortex, and so forth.

Many times other systems of a different origin are also affected, and then one speaks of pleiotropism, examples of which are the Bardet-Biedl syndrome which includes atypical retinitis pigmentosa with adiposogenital dystrophy, polydactyly, and mental deficiency. One can see at times in the same family several members who show the complete syndrome and some who show only isolated symptoms.

This concept has been amplified by Riddell under the name of "multiple heredity." This includes various pathologic genes in the same pedigree. Examples of this are retinitis pigmentosa with cataract and idiocy. It seems that the pathologic genes which come together do not combine with each other but retain their independence.

Dr. Marin-Amat called attention to two things: (1) The simultaneous occurrence of the macular lesion and of those corresponding to the pigmentary degeneration of the retina; (2) total atrophy of the papilla, early in the disease. With reference to the simultaneous occurrence of the lesions in the macula and in the rest of the retina, he mentioned the case of a boy with amaurosis of familial idiocy, in whom were present, at the same time, atrophy of the papilla and incipient nystagmus.

Joseph I. Pascal,
Translator.

CHICAGO OPHTHALMOLOGICAL SOCIETY

May 18, 1953

WILLIAM F. HUGHES, JR., *president*

The clinical meeting was presented by the Department of Ophthalmology, University of Chicago, Dr. Duval Jaros, presiding.

CROUZON'S DISEASE

DR. SHELDON ZINN presented R. K., first seen at Bobs Roberts Hospital in August, 1949, at the age of 10 months. She was brought in because of abnormal structural features which included protruding eyes, a peculiarly shaped skull, high forehead, stiff elbows, and a pilonidal dimple. The family history indicated that a great grandfather was said to have a tower skull. The prenatal history was noncontributory.

It was noted at birth that the child had proptosis, an abnormally shaped skull, and a pilonidal cyst. A diagnosis of Crouzon's disease was made at another hospital at the age of one month. There was no history of signs of increased intracranial pressure. The head seemed to become more malformed with growth but the eyes did not become more proptosed.

Examination at this time revealed a baby girl who looked like a frog—the eyes markedly exophthalmic with an associated exotropia. The interpupillary distance measured 75 mm. The bridge of the nose was flat and sunken; the jaw seemed protruding. In addition to the cranial features, complete ankylosis of both elbows and a pilonidal cyst were found.

X-ray studies indicated pronounced oxycephaly of the skull with a "pounded copper" appearance in the posterior half of the cranial vault.

In May, 1951, it was noted that the optic discs were blurred at the edges but a true papilledema was not noted. In July, 1952, at the age of three years, a diagnosis of early papilledema was made. Because of the increased intracranial pressure, ventriculograms and surgery were advised, and in September, 1952, a morcellation procedure was successfully performed.

In April, 1953, the significant ocular findings were exophthalmos measuring 27 mm. in the right eye and 24 mm. in the left. The disc margins appeared obliterated although no dioptric elevation was noted. The patient was 20 prism diopters exotropic. Intelligence was considerably retarded. Vision: R.E., 5/15; L.E., 5/30.

The case is presented as one of dysostosis craniofacialis or Crouzon's disease. This rare deformity is characterized by exophthalmos, atrophy of the maxillas, prognathism, enlargement of the nasal bones, optic atrophy, and bony abnormalities in the region of the superior longitudinal sinus and bregma.

TUBEROUS SCLEROSIS

DR. PETER M. HAYDEN said that A. W., a white woman, aged 22 years, was first seen at Billings Hospital in May, 1948, complaining of a rash on her face which had been present since the age of two and one-half years, and several fainting spells the previous year.

Dermatologic examination revealed small erythematous papules on the nose, cheeks, forehead, and chin which, on biopsy, proved to be sebaceous adenoma of the Pringle type. Electro-encephalogram, pneumo-encephalogram, and cerebrospinal fluid were within normal limits and psychometric evaluation demonstrated average intelligence.

Ophthalmologic study indicated a beaded, round, grayish-white mass, elevated 1.5 diopters, and one-third disc diopter across, arising from the nasal quadrant of the right disc. Contiguous with the left disc, inferotemporally, was a globular, grayish-white, beaded mass of crystalline appearance, elevated three diopters and one disc diameter across. Except for a four prism diopter intermittent alternating exotropia and a sug-

gestion of exophthalmos, the eye examination was negative.

The lesions of the face were treated with moderate success with CO_2 "snow" applied at frequent intervals over the subsequent two and one-half years.

The signs described are compatible with a diagnosis of tuberous sclerosis, known eponymically as Bourneville's disease. It is usually seen in early childhood, and the complete syndrome is characterized by epilepsy, mental retardation, skin changes, and fundus changes. Although adenoma sebaceum is the most consistent dermatologic finding, cafe-au-lait spots, vitiligo, and fibromas are often observed. The fundus changes, located in the posterior pole, range from flat phakomas to the typical elevated "mulberry" tumor. The course of the disease is slowly progressive, and there is no known treatment.

Richard C. Gamble,
Recording Secretary.

Ophthalmic Miniature

Samuel Pepys laments the failure of his eyesight:

11/4/1667: From the exchange I took a coach and went to Turlington, the great spectacle maker, for advice, who dissuades me from using old spectacles, but rather young ones, but do tell me that nothing can wrong my eyes more than for me to use reading glasses which do magnify much.

7/31/1668: The month ended mighty sadly for me, my eyes being now past all use almost, and I am mighty hot upon trying the late printed experiment with paper tubes.

5/8/1669: Up and to the office, and there comes Lead to me, and at last my vizards are done, and glasses got to put in and out as I will; and I think I have brought it to the utmost, both for easiness and for using and benefit, that I can. And so I paid him 15s. for what he hath done now last, in the finishing them, and they, I hope, will do me a great deal of ease.

5/31/1669: And thus ends all that I doubt that I shall ever be able to do with my own eyes in the keeping of my Journal.

(The tubes shut out extraneous light to lessen the blur but it is evident Pepys needed something more than simple plus lenses and shutting out extraneous light. R.I.L.)

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XVII INTERNATIONAL CONGRESS OF OPHTHALMOLOGY MONTREAL—NEW YORK

Almost a century is a long history for the continuity of international congresses—the longest history in medicine or any of its branches; but this XVII International Congress of Ophthalmology, held by the two great English-speaking countries of North America, has been the biggest and the best in the entire series.

To organize a congress of this type in this era of rapid and easy travel, when the world is spanned in days instead of weeks, is a tremendous undertaking, for the whole ophthalmologic world gathers for inspiration, for knowledge, and for relaxation. And so from the five continents we came, some of us eager to renew old friendships, others of us full of anticipation to see the wonders of the new world; and all of us were received

into a wealth of human kindness and warmth of hospitality that none of us will ever forget.

From the scientific point of view the congress was an outstanding success. Ample fare was offered to the hungriest; and those of us who did not know it before at first-hand were impressed by the drive and massive proportions of North American ophthalmology; it has indeed become a leading influence in the advancement of our science. Nor were contributions of great merit and on all aspects of ophthalmology lacking from the Old World. The immense amount of material contained in the long list of papers will make the *Acta* of unusual interest and importance. The exhibits and demonstrations had, of course, to be seen to be appreciated, but they—as well as the motion pictures and televised operations, which were shown on an impressive scale—rounded off an intellectual banquet that will take long to digest and assimilate.

But if the scientific aspect of the congress was good, the social aspect was superb. The atmosphere was nicely variegated, from the academic surroundings of the Universities of Montreal and McGill to the city-within-a-city of the Waldorf-Astoria. We were received by the open arms of the Canadians, passed across a political boundary that did not exist, and were taken right into the great, warm heart of the United States, into a wealth of kindly and cheerful hospitality, the equal of which exists nowhere in this world. All the time among friends and all the time at home, we had an experience which none of us can forget.

To discuss ophthalmology is good; to meet each other is better; to realize that, no matter what language we speak or what country we come from, how nice we all are and how much we have in common, in sympathy and outlook, is best of all. To our North American colleagues, and particularly those on whom the great task of organization fell, the whole ophthalmologic world owes an immense debt which can never be repaid materially but which will pay spiritual dividends in abundance.

Stewart Duke-Elder.

CHLORPROMAZINE

A NEW ANTI-EMETIC AGENT

The problem of the vomiting patient has plagued the ophthalmic surgeon for many years. Vomiting in the early postoperative period has been held responsible for a number of complications of eye surgery including hemorrhage, infection, wound rupture, and iris prolapse. Particularly dreaded is the distressing occurrence of emesis during such intraocular procedures as cataract extraction. Besides the resultant dangers of vitreous loss and infection, this hazard induces enormous emotional stress in operating-room personnel. Usually of less consequence, but still adding greatly to the patient's discomfort, is the high incidence of postoperative vomiting following strabismus surgery in children under ether anesthesia.

Ophthalmic surgeons should be made cognizant of the availability of a new agent, chlorpromazine, which permits the virtual elimination of the hazards of operative and postoperative emesis. In addition to its remarkable anti-emetic activity, this drug possesses the useful property of augmenting the effects of anesthetics, sedatives, narcotics, and curare in patients undergoing ocular surgery.

Chlorpromazine (called Thorazine in this country and Largactil in France and Canada) is a central nervous system depressant with diverse pharmacologic properties, acting principally on hypothalamic areas. It has been demonstrated to prevent apomorphine-induced emesis in dogs, to alter conditioned reflexes in rats, to influence body temperature and blood pressure, to sedate without inducing hypnosis, and to aid in the management of unco-operative and agitated psychiatric patients.

The administration of chlorpromazine to patients has been demonstrated repeatedly to prevent or to control the vomiting associated with a wide variety of conditions. It has been used with considerable success in vomiting during pregnancy. Promising results have also been reported in the persistent and refractory emesis associated with uremia, cholecystitis, gastro-enteritis, hepatitis, labyrinthitis, diabetic acidosis, intestinal obstruction, leukemia, carcinomatosis, and increased intracranial pressure. It has also proved effective in the prevention and treatment of nausea and vomiting induced by the administration of nitrogen mustards, urethane, radiation, antabuse, antibiotics (for example, aureomycin and terramycin), opiates, and so forth. Even in desperately ill patients with impaired renal or hepatic function, the drug has been administered safely and without significant toxicity. Side effects reported include drowsiness and dizziness with occasional transient postural hypotension. Jaundice, which mimics the obstructive type of jaundice, and skin rashes have appeared in a few patients, usually after two or three weeks' therapy, but have subsided when the drug was discontinued.

Laborit and co-workers in France have demonstrated the value of chlorpromazine in surgery. Its use facilitated anesthesia, decreased the patient's need for postoperative narcotics, and prevented postoperative vomiting. These properties suggested a trial of this drug in eye surgery.

The efficacy of the prophylactic and therapeutic uses of this agent in ophthalmology have been confirmed recently in Saint Louis by members of the Department of Ophthalmology, Washington University School of Medicine. In these studies the therapeutic administration of Thorazine to adults in doses of 50 mg. intramuscularly followed by 25 mg. orally every four to six hours alleviated dramatically all instances of nausea and vomiting following surgery on the eye. The use of this anti-emetic agent also proved of enormous value in patients with acute glaucomas, in traumatic injuries, and in surgical emergencies in intoxicated patients. Prompt cessation of vomiting permitted restoration of electrolyte balance and better hydration. It also afforded the opportunity for administration of oral medications (antibiotics, sedatives, Diamox, and so forth). When given prophylactically to children subjected to ether anesthesia, the drug prevented vomiting almost completely. The routine prophylactic use of chlorpromazine in all intraocular surgery in the preoperative and early postoperative period (oral doses of 25 to 50 mg. every four to six hours for adults) avoided the hazards of operative and postoperative vomiting in most striking fashion. Furthermore, it permitted the use of smaller doses of preoperative sedatives and postoperative narcotics, and provided for the surgeon a more co-operative patient.

It is apparent from these observations that chlorpromazine possesses properties that can prove of great value to the ophthalmologist. The routine use of this agent in the preparation and care of the patient undergoing ocular surgery not only prevents vomiting, but results in cheerful, more relaxed patients and ophthalmic surgeons.

Bernard Becker.

CORRESPONDENCE

Postgraduate quiz

Editor,
American Journal of Ophthalmology:

While going through my papers the other day, I noticed the following case history which was written by Dr. Richard G. Scobee as a quiz for the postgraduate students in 1948.

It seems to me that this is well worth publishing as an example of a method of examination, but especially of the wit and humor of our late associate.

(Signed) Paul W. Miles,
Saint Louis, Missouri

Ophthalmology Quiz

March 19, 1948

Case history

John Z., 54-year-old Bedouin, presented himself at the Eye Clinic of the American Hospital in Beirut, seeking eye glasses. It may be significant that he was *led* in by two men, one a first cousin who was seemingly in perfect health and the other a second cousin with some strange malady.

Although the patient spoke no English, some history was obtained through interpreters. At birth, his skin had been a dusky blue color and was still definitely pigmented. The patient had never been in good health at any time during his life. His vision, however, had seemed normal until two weeks before when it began to fail rather rapidly and he reached the point where he was unable to read the *Beirut Daily Bugle*. Interestingly enough, he had never been able to read the *London Times*. There were no other specific complaints except shortness of breath after getting out of bed in the mornings.

The vision was 20/400 in the right eye and no light perception in the left. External examination revealed a left exotropia and left hypotropia, both quite extreme, and the left eye appeared to be fixed in this down-and-out position, irrespective of the direction of gaze of the right eye. The pupils were round and regular but unequal, the right pupil measuring three mm. while the left measured five mm. The right pupil reacted to light directly but not consensually and the pupil of the left eye was fixed. There was a monocular right horizontal nystagmus.

Manifest refraction (retinoscopy) revealed:

O.D., +1.5D. sph. ⌒ +0.75D. cyl. ax. 82° = 20/300; O.S., +8.0D. sph. ⌒ −6.0D. cyl. ax. 75° = no light perception.

Tension to fingers: O.D., normal; O.S., very hard.

Visual field studies were attempted but could not be made successfully.

Slitlamp: O.D., entirely normal; O.S., roughened, cloudy cornea; the iris structure was very coarse and of a peculiar color; there was what appeared to be a vitreous hemorrhage bulging through the fixed pupil; no blood vessels were seen on the iris.

Fundi: O.S., no red reflex; could not be observed. O.D., no opacities in the media; disc reddened, no cup, margins very blurred elevated 2.0D. Veins somewhat engorged, as were the arteries. Scattered over the fundus in the region around the disc were several roughly circular small reddish spots, some with white centers. No other significant finding. There was moderate arteriosclerosis compatible with the age. It was felt that something mysterious was wrong with the patient and he was hospitalized for careful laboratory study. The findings were as follows:

Blood: Rbc—2,800,000. Hb—7 gm. percent
Wbc—4,000 c moderate shift to left.
Sed. rate—39 mm./hr. (corrected for anemia)
Platelets—200,000
Bleeding time—5 min. (Ivy method)
Eagle—negative

Urine: Dark brown, growing darker on standing.
Sp.Gr.—1.030 pH—8.5
Sediment—5 rbc/hpf., 5 wbc/hpf., few granular casts, also cystein crystals
No bacteria on stained smear
Sugar—trace
Albumin—trace

Blood chemistry:
NPN—35 mg. percent
Sugar—120 mg. percent
Total proteins—4 gm.
A/G ratio—2/1
Phosphatase—4 Bodansky units
CO_2 combining power—60 vol. percent
Total blood fats—2.5 gm./100 cc.

Blood cultures:
Negative on two occasions
Staph. aureus on one occasion
Unidentified gram-negative bacilli and gram-negative diplococci as well as Staph. albus on one occasion

Agglutination tests:
Typhoid—positive in 1/10 dilution
Paratyph. A—positive in 1/5 dilution
Tularense—positive in 1/20 dilution
Brucella—positive in 1/20 dilution

B proteus (X19)—positive in 1/2 dilution (OXK not available)
Cold agglutination titer—1/20
Paul Bunnell test—negative
Complement fixation test positive for nonhemolytic streptococcus

Miscellaneous tests:
Klein (not Kline) test—negative
Pels-Macht test—no rootlets of Lupinus albus available. No effect on cottonwood
Gothlin index—14

Unfortunately the patient died before further essential laboratory procedures could be carried out. The accompanying cousins refused to permit an autopsy but instead took the patient, folded their tents, and silently stole away in the night, leaving only a bottle of eyedrops (Argyrol) and a bottle of Scobee's capsules (practically untouched).

Please give the most likely diagnosis for: (1) Right eye; (2) left eye; (3) systemic condition.

BOOK REVIEWS

LYLE AND JACKSON'S PRACTICAL ORTHOPTICS IN THE TREATMENT OF SQUINT (and other anomalies of binocular vision). Revised by T. Keith Lyle, assisted by Marianne Walker. With 195 illustrations, including three colored plates. Philadelphia and Toronto, The Blakiston Company, 1953, fourth edition. Price: $12.00.

The "Lyle and Jackson" has now appeared in a fourth edition: ample evidence of its vitality and usefulness. A comparison of the third and fourth editions shows that the revision has been a very thorough one. The text has been increased by 100 pages and the number of illustrations by 44. Many of the old illustrations have been replaced by new, more up-to-date ones.

The text has been both amplified and rearranged. The chapter on orthoptic instruments has been rewritten and the technique of examination included in the same chapter. A chapter on ocular neuroses and a chapter on nystagmus have been added; aniseikonia is briefly discussed. Throughout, the text has been reorganized, enlarged, and corrected, all of which has materially enhanced the value of the book. The largest addition has been made to the chapters on paralytic strabismus which now occupy 103 pages, as compared to 46 pages in the previous edition.

It is not possible to make a detailed critical evaluation of this book within the framework of a brief note, but it is interesting to ponder the differences in the development which orthoptics has taken in England and America. Using the "Lyle and Jackson" as the authoritative expression of British orthoptic thought and comparing it with an imaginary American textbook, endorsed by leading authorities in the field, many differences are evident. The American textbook would contain a much more detailed discussion of visual physiology, space sense, and the physiology of the ocular motility. Child psychology and the learning process as related to orthoptics (which received no mention by Lyle and Jackson) would be dealt with at some length. Such subjects as the accommodation-convergence relationship would be much more fully treated. Accommodative strabismus and its treatment would be more prominent, and the same would be true of convergence insufficiency and allied anomalies. On the other hand, operative treatment and paralytic strabismus would receive much less space than given to them by Lyle and Jackson. In general, the American textbook would not place the emphasis so much on the ophthalmic diagnosis of the motor anomaly as on the orthoptic diagnosis and above all on the treatment.

The "American textbook" referred to does not, however, exist as yet and we must be grateful to Dr. Lyle and his collaborators that they continue to furnish us with their ever-improving manual on orthoptics.

Hermann M. Burian.

ORBITAL PLASTIC. By Dr. István Csapody. Budapest, Akadémia Kiado, 1953. 102

pages, 71 illustrations, 7 tables, bibliography. Price: Not listed.

In *Orbital Plastic,* Csapody describes his method of surgical correction of the generally contracted socket. The author bases this monograph on his experience with a total of 151 sockets—operated either by himself or his disciples under his supervision. This is indeed an admirable series of cases that is difficult to rival. The first operation was performed 25 years ago. After some experimenting, Csapody perfected the definitive routine 15 years ago and he has followed it since that time.

Because the crux of all orbital plastics is the shrinkage of the transplants, Csapody felt that he had to develop a technique to avoid this pitfall. This he did in a truly ingenious manner:

While a single flap would cause shrinkage toward the center of the cavity, a zig-zag incision at the apex of such a flap would divide it into two separate portions. Then the centrifugal tendency of each of the two parts of the flap to shrink toward the lid margin would counteract the centropedal tendency of the entire flap to shrink toward the center of the cavity. Such a method proved to be more practical than the use of two separate flaps—one for the upper and one for the lower lid—in a two-stage operation, as practiced at first.

In brief, the method that Csapody used in performing the operation is as follows:

A uniform pattern made of metal serves for all cases, since it proved entirely practical to use prostheses of one standard size in every instance. The author uses a free full-thickness graft from the inside of the upper arm. This graft is sutured to a mold of his own construction. It consists of two spoon-shaped hemispheres that are jointed, enabling him to separate them to suit the individual case.

The tremendous advantage of this procedure is that it is a typical method to correct a deformity which heretofore had been corrected by atypical methods. Since the general consensus is that the results of most orbital plastics are unsatisfactory, most surgeons try to avoid them. Csapody describes in great detail not only the operation itself, but also the protracted and important after-care. Nevertheless, I feel that a word of caution is in order: even with this step-by-step description, the operation appears rather complicated and should be reserved for surgeons who are well versed in plastic procedures.

The book contains a number of typical case histories described in great detail, as well as a summary of all cases which the author has seen. The schematic illustrations are excellent and give a vivid idea about the principle of the method. Reproductions of actual photographs give the impression that it is rather an understatement for the author to speak of "excellent" end results.

Stefan Van Wien.

Biology of the Lens. (In French.) By Jean Nordmann. Paris, Masson et Cie, 1954. 730 pages, 169 figures, 39 tables. Price: Not listed.

In his *Biologie du Cristallin* Jean Nordmann has written a worthy companion to its 21 predecessors in the series of publications of the Société française d'Ophtalmologie. This monograph exhibits all the excellence which we have come to expect in this fine series of books. The entire knowledge of the lens is systematically presented, not merely collected and recorded but evaluated so that the data may be related to a meaningful pattern. The reader of any section will find answers to all reasonable questions, including many which it had not occurred to this reviewer to ask. At the end of each section, there is a section entitled "Conclusion" which contains a brief summary of results of the investigations and an adequate bibliography.

The normal lens is described in the first 290 large pages in eight categories: Embryology, anatomy, physics, chemistry, physi-

cal chemistry, static physiology, dynamic physiology, and immunology.

In the section on anatomy, the biomicroscopic landmarks and their characteristics at different ages are discussed no less adequately than the gross anatomy and histology.

The section on physics begins with a discussion of weight, specific weight, and temperature. The temperature is not only from 1°C. to 2°C. lower than body temperature but also varies with the outside temperature. The larger part of this portion of the book deals with optic, mechanical, and micellary characteristics.

Chemistry deals with the chemical constituents of the lens, and static physiology with their role in metabolic changes. In physical chemistry, pH, iso-electric point, oxidation-reduction, equilibrium, and osmotic pressure are dealt with.

The section on dynamic physiology deals with the position of the lens in repose, changes in the lens during accommodation, the factor acting on the lens in the course of accommodation, the mechanism of accommodation, and the process of presbyopia.

In the chapter on immunology, the author discusses the sensitivity of normal man to heterologous, homologous, and autologous lens protein.

In the larger part of the book the author discusses the pathologic lens. In an introductory section of 40 pages the noncataractous anomalies are described: the congenital anomilies, aphakia, microphakia and spherophakia, coloboma, lenticonus, and anomalies of position, and such acquired anomalies as those of capsule and zonula, of form, of the bands of discontinuity, and of refraction. Separate sections of one page each are devoted to discussion of infection, overabundance of inorganic and organic substances, such as occurs in siderosis and chalcosis, and neoplasia. Although cancer of the lens has not been observed in man, it has been shown that the epithelium of the lens can be subject to malignant transformation.

The remainder of the book, almost 400 pages, is a monograph on cataract. All the congenital and acquired forms of cataract are described. The pathologic anatomy, heredity, physics, chemistry, physical chemistry, and physiology of the cataractous lens are discussed thoroughly. In further sections, the author analyzes the relation of immunology to cataract, its pathogenesis, and the various forms of medical therapy.

F. H. Haessler.

ORMONE CORTICOTROPO E CORTISONE IN OFTALMOLOGIA. By G. Calamandrei, L. Ferrata, and L. Montanari. Preface by Prof. Donato Cattaneo. Pisa, Industrie Grafiche V. L. Lischi & Figli, 1953. 166 pages. Price: 1,500 lire.

Having previously published their own observations on the effects of cortisone and ACTH, the authors—two of them connected with the University Eye Clinic of Milan and one with the Institute of Biologic Chemistry of the Bologna University—proceeded to summarize these experiences, and the reports of other authors, in a comprehensive monograph. By accenting the most important facts and by critical evaluation, the danger of simple compilation was avoided.

The introduction includes the history of the detection of these hormones, their chemical qualities, and their biologic actions. The ophthalmologic part is divided into descriptions of methods of administration, topographic and clinicopathologic chapters, in which the authors discuss the action of these substances on blood vessels, on the blood-aqueous barrier, on the vegetative nervous system, granulation tissue, epithelium and endothelium, and vascularization.

The effects on experimental infections (tuberculosis, various virus and other infections) are discussed from the standpoint of the ophthalmologist, as well as from a general pathologic viewpoint. Thirty-five pages of references show the many contributions of Italian researchers but give full credit to other European and American authors.

K. W. Ascher.

THE GEOMETRY OF BINOCULAR SPACE PERCEPTION. By L. H. Hardy, M.D., G. Rand, Ph.D., M. C. Rittler, B.A., A. A. Blank, Ph.D., and P. Boeder, Ph.D., Elizabeth, New Jersey, J. Schiller, 1953. Paperbound, 68 pages, 30 figures, bibliography.

This research is probably the last work sponsored by Le Grand H. Hardy, the principal investigator, whose illustrious career was recently untimely terminated. This important report represents the work of five years and presents for the first time extensive experimental results which strongly support the Luneburg theory. The intricate mathematical analyses, initiated by the late Rudolph K. Luneburg himself, were continued by A. A. Blank with the co-operation of P. Boeder, H. Coxeter, C. Campbell, B. Altschuler, A. Stein, C. H. Graham, and W. Berry. All experiments were carried out in a darkroom. The observer's head was fixed but the eyes were allowed to vary fixation at will until a stable perception of the geometry of the situation was achieved. Given a pair of light points it was possible to arrange others along a curve which was perceived as the extended straight line joining the points. Analysis shows that the binocular visual space is one of the riemannian spaces of constant gaussian curvature and has constant characteristics for a given observer. Some practical deductions result from the increased understanding of space judgments in situations providing reduced clues. "For example, it may be suggested that parallel rows of guide lights be set up along all airplane runways at a uniform standard separation of the rows and at a uniform standard spacing of the lights, so that a pilot landing at night can rely on facing the same situation each time he lands at any field and on any runway." Evidently such considerations motivated the Office of Naval Research to support this investigation which was done at the Institute of Ophthalmology of Columbia University. James E. Lebensohn.

A TREATISE ON CONIC SECTIONS. By George Salmon, D.D., F.R.S. New York, Chelsea Publishing Co., 6th edition, 1954. 399 pages. Price: Paper-bound, $1.94; cloth, $3.25.

This text by the Regius Professor of Divinity in the University of Dublin contains a comprehensive account of the most important modern algebraic and geometric methods including all essential parts of the theory of analytic geometry. Much of the research in ophthalmology, past and present, cannot be followed adequately without a good mathematical background such as is obtainable from this basic text. The publishing company is to be commended in following the European practice of making such technical publications available in inexpensive paperbound editions. James E. Lebensohn.

ABSTRACT DEPARTMENT

EDITED BY DR. F. HERBERT HAESSLER

Abstracts are classified under the divisions listed below. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is mentioned only in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. Anatomy, embryology, and comparative ophthalmology
2. General pathology, bacteriology, immunology
3. Vegetative physiology, biochemistry, pharmacology, toxicology
4. Physiologic optics, refraction, color vision
5. Diagnosis and therapy
6. Ocular motility
7. Conjunctiva, cornea, sclera
8. Uvea, sympathetic disease, aqueous
9. Glaucoma and ocular tension
10. Crystalline lens
11. Retina and vitreous
12. Optic nerve and chiasm
13. Neuro-ophthalmology
14. Eyeball, orbit, sinuses
15. Eyelids, lacrimal apparatus
16. Tumors
17. Injuries
18. Systemic disease and parasites
19. Congenital deformities, heredity
20. Hygiene, sociology, education, and history

3

VEGETATIVE PHYSIOLOGY, BIOCHEMISTRY, PHARMACOLOGY, TOXICOLOGY

Venturi, G., and Preti, A. **The retinal arterial pressure and the ocular tension during and after surgical operations.** Ann. di ottal. e clin. ocul. **80**:83-100, 1954.

Blood pressure of the retinal arteries and ocular tension were measured in 32 patients who had general surgical operations and compared with the general blood pressure. The fall in retinal blood pressure was more marked than that in the humeral artery while the changes in ocular tension more or less followed the general blood pressure. The retinal arteries showed particularly a reduction of the diastolic values, which indicates that they participated in the vasomotor phenomena demonstrated in other circulatory areas during surgical trauma. In view of the functional relations between the retinal and cerebral arteries it is suggested that these conclusions can also be applied to the cerebral circulation.

John J. Stern.

Wootton, J. F., Young, R. G., and Williams, H. H. **Biochemistry of the eye. 1. Chromatographic investigation of free amino acid constituents of vitreous humor.** A.M.A. Arch. Ophth. **51**:589-592, May, 1954.

The study was made on fresh bovine eyes. The vitreous was separated and filtered by gravity. The amino acids were extracted chemically. The analyses reported in this investigation support the view that the amino acids present in the vitreous humor represent an ultrafiltrate of blood, although the relative concentration tends to be lower. Some qualitative and quantitative differences were found in the amino acid patterns of vitreous filtrates prepared for chromatographic analysis by different methods. In addition to 17 known amino acids, the presence of several as yet unidentified ninhydrin-reactive substances in the vitreous has been revealed.

G. S. Tyner.

Yasukura, K. **Relationship between ascorbic acid and dinitrophenol cataract.** Acta Soc. Ophth. Japan **58**:570-573, July, 1954.

In contrast to Tainer's report (Arch. Ophth. 20:30, 1938), Yasukura was able to produce cataract in guinea pigs with α-2-4-dinitrophenol. The agent was given in 10-mg. doses daily by mouth. Seven of

the ten animals died within one month without cataract development. In one other animal cataract developed after 36 days. The last two animals were free of cataract even after 105 days administration of the agent. When the animals were fed without vitamin C, the cataract development occurred earlier and in a higher percentage. The author further states that there is an excretion of cataract-developing substance in the urine of rabbits to whom dinitrophenol is administered. An ether extract of such urine can produce cataract constantly in guinea pigs with vitamin C deficiency when introduced intraperitoneally. (5 tables, 13 references)

Yukihiko Mitsui.

Young, R. G., and Williams, H. H. **Biochemistry of the eye. II. Gelatinous protein of vitreous body.** A.M.A. Arch. Ophth. **51**:593-595, May, 1954.

The vitreous was obtained from fresh bovine eyes. The material obtained after washing showed an amino acid pattern similar to that of gelatin. G. S. Tyner.

4

PHYSIOLOGIC OPTICS, REFRACTION, COLOR VISION

Alpern, Mathew. **Spatial range of metacontrast.** Am. J. Optometry **31**:363-369, July, 1954.

Additional pattern in the visual field at the moment of a brief near threshold stimulus can change the apparent stimulus brightness. The metacontrast was studied with various flash luminance from 1 to 1,000 ft.-L. There was no increase in the spatial range with greater intensity.

Paul W. Miles.

Bartlett, N. R., and Macleod, S. **Effect of flash and field luminance upon human reaction time.** J. Optic. Soc. America **44**: 306-311, April, 1954.

The interval of time after a flash of light until a person could press a microswitch was measured for both foveal and peripheral stimuli. Tests were made on both dark and light adapted eyes with various intensities of visual field. Resulting curves showed that low intensities produced a longer latency before response.

Paul W. Miles.

Bartlett, N. R., and Macleod, S. **Human reaction time during dark adaptation.** J. Optic. Soc. America **44**:374-379, May, 1954.

As the intensity of stimulus was reduced, the reaction time increased. In dark adaptation the relative effect of a fixed stimulus became greater. Typical dark adaptation curves, however, could not be produced by this method.

Paul W. Miles.

Bradford, R. T., and Lawron, L. J., Jr. **Clinical evaluation of the Rodenstock refractometer.** A.M.A. Arch. Ophth. **51**: 695-700, May, 1954.

After a preliminary trial of the instrument in several hundred refractions, a series of 70 patients from 9 to 38 years of age with clear media and no ocular abnormality were studied under cycloplegia. Each patient was refracted by streak retinoscopy, subjective trial and with the Rodenstock instrument. A further comparison was made with and without cycloplegia. The Rodenstock instrument was superior to retinoscopy in patients with scissors movements. The instrument was inaccurate in 25 percent of patients without cycloplegia because of excessive accommodation. In patients without cycloplegia and with small pupils (not less than 1.5 mm.), the instrument was accurate. Cloudy media interfered more with the refractometer than with retinoscopy. Much less skill was required to successfully operate the instrument than to refract by ordinary methods.

G. S. Tyner.

Choyce, D. P. **Glass contact lenses. Effects of ventilation.** Brit. J. Ophth. **38**: 100-114, Feb., 1954.

The wearing of ventilated contact lenses by a series of subjects was investigated and compared to a previous analysis of the use of nonventilated glass contact lenses. The fitting had been performed by one small group and is discussed in detail. Suction between lens and globe is eliminated and air bubbles are always present. It was found that considerably fewer patients have given up wearing the ventilated lenses, and that bubbles are much less of a nuisance. In general the performance of the ventilated lenses was better, but not strikingly so.

Orwyn H. Ellis.

Dzn, H. de Lange. **Relation between critical flicker-frequency and a set of low-frequency characteristics of the eye.** J. Optic. Soc. America **44**:380-389, May, 1954.

This article treats the vision of flicker mathematically and the eye is considered a low pass filter which flattens a sine wave. Paul W. Miles.

Gloster, J. **Factors influencing the visual judgment of the vertical direction.** Tr. Ophth. Soc. U. Kingdom **73**:421-433, 1953.

The author discusses the A and E phenomena in which apparent tilting of a vertical line in a darkened room is caused by tilting the head. Aubert in 1861 made the interesting observation that in a completely darkened room a luminous line appears tilted when observed with the head inclined to one side. As soon as the room is illuminated and other objects become visible the apparent tilt vanishes. Aubert's observation was called the A phenomenon. Nagel and others used a line which could be rotated about an axis in a completely darkened room until it was judged to be vertical and this has been called the E phenomenon by Müller. Typically the A phenomenon is found when the angle of head tilt is large while the E phenomenon accompanies small tilts.

In the author's experiments the observer estimated the vertical in a dark room, 1. with the head upright, 2. with the head tilted 30°, and 3. when lying on his side. A biting-board was used to hold the head steady at a known angle of tilt. Forty consecutive estimations of the vertical were made, the entire experiment lasting about 20 minutes. Few errors were made when the observer was upright. The errors were greater when the head was tilted and they were very large when the observer lay on his side. The observation of the A phenomenon did not seem to be related to the time spent in darkness but to the time spent in the tilted position, which suggests that it is due to some change in the postural contribution to the perceptual process.

The test for the E phenomenon was carried out first after the observer had been in darkness for 20 minutes and then without a preliminary period in darkness. The room was illuminated between successive estimations of the vertical. An analysis of the data makes clear the difficulty of interpretation.

The central mechanism concerned in visual perception of the vertical is extremely complex and few clinical conditions have been reported in which it is disturbed. Beulah Cushman.

Hill, Armin J. **Wide screen and stereoscopic pictures.** Am. J. Optometry **31**:342-362, July, 1954.

This is a mathematical analysis of binocular vision based on the subjective appearance of stereoscopic motion pictures of objects at various distances with the two camera lenses at various separations. The work has obvious practical value, but is important to binocular theory as well. In the movies, the accommodation should

remain at the screen distance while vergence may vary from infinity to a few feet. The limits of convergence and of divergence tolerated by audiences is surprisingly small. For "giant" close-up portraits, the stereoscopic disparity must be reduced by bringing the two lenses closer together. Paul W. Miles.

Littmann, H. **Corneal astigmatism of a previously undescribed type.** Arch. f. Ophth. **154**:603-616, 1954.

Corneal astigmatism is usually determined with the help of the Javal-Schiötz ophthalmometer. The refracting vertex powers of the cornea are measured in two meridians perpendicular to each other and their difference is a measure of the corneal astigmatism. Different values for other points of the cornea are obtained when the instrument is directed at the center of the corneal curvature but not at the midpoint of the pupil. It is the purpose of this paper to emphasize the different degrees of corneal astigmatism in different corneal areas. At the same time the importance of the correct aiming of the ophthalmometer at the pupillary center becomes evident. (12 figures, 2 references) Ernst Schmerl.

Lyle, T. Keith. **The importance of orthoptic investigation before contact lens fitting in unilateral aphakia.** Tr. Ophth. Soc. U. Kingdom **73**:387-398, 1953.

The author describes 35 unilateral aphakic patients, 26 of whom obtained binocular single vision and nine who remained functionally uninocular with the use of contact lenses. Treatment in children should be carried out early because suppression develops so rapidly. A forecast of the ultimate result can be determined by an orthoptic investigation.
Beulah Cushman.

McLaughlin, S. C., Jr., **An automatic recording visual adaptometer.** J. Optic. Soc. America **44**:312-314, April, 1954.

A new instrument for the clinical measurement of dark adaptation requires little attention from a technician. The target is exposed intermittently 0.1 second every two seconds. Paul W. Miles.

Monjé, M. and Offermann, R., **The sensitivity of the retina in anomalies of refraction.** Arch. f. Ophth. **155**:63-78, 1954.

The authors apply a modified perimetry with special test objects to studies of the retinal sensitivity. In emmetropia the sensitivity to size and brightness of an object is highest for the fovea, declines by 50 percent for the 10° circle of the visual field and by 90 percent for the 40° circle. A myopia of −2D shows a slightly diminished sensitivity of the fovea, and at −16D the fovea appears less sensitive than the nasal area of the retina. Similar phenomena are seen in hyperopia, although they are somewhat less pronounced. With higher errors of refraction the blind spot becomes more and more difficult to demonstrate. (4 figures, 1 table, 27 references) Ernst Schmerl.

Piper, H. F., and Rosehr, K. **Examination of the sensory-motor function of the visual apparatus.** Arch. f. Ophth. **155**:35-62, 1954.

The purpose of the present study is to combine tests for light adaptation with those for visual acuity and muscular response. The authors use a special instrument called a projection adaptometer which offers fixed and moving objects. With the help of this instrument they tried to determine the threshold of sensitivity to light, the threshold of perception of movements, the threshold of nystagmus, and the beginning of the acute vision. From their findings they reach the conclusion that vision shows functions of different qualities with respect to environment. They feel that there exist higher

functions with a fixed relationship to the visible environment, and lower functions more losely connected with it. The ocular motility permits a certain width of perception of the resting and the moving environment. The recognition of structures and movements is ascribed to the higher visual functions, while a more general awareness of the environment might be considered a quality of a lower degree. The original paper should be consulted by those interested in a theoretical discussion of the involved problems. (11 figures, 1 table, 32 references) Ernst Schmerl.

Purvis, Victor. **Some consideration upon the value of early visual acuity tests in the school child.** Tr. Ophth. Soc. U. Kingdom **73**:415-420, 1953.

Of 403 school children tested who were five years of age, 11 had already received attention, two for high myopia and nine for strabismus. Of the remaining 392 all but 20 had normal visual acuity of 6/6 in each eye. Seventeen of these 20 defective children had refractive errors and three were noncooperative. In eight of them the vision was 6/20 or less. The visual acuity tests on five-year-old children are reliable and all children should be examined on entry into school. Beulah Cushman.

Ridley, Frederick. **Contact lenses in unilateral aphakia.** Tr. Ophth. Soc. U. Kingdom **73**:373-386, 1953.

Of 108 patients with unilateral aphakia 84 were given contact lenses. These were divided into traumatic and nontraumatic. The factors of greatest significance in assessing the likelihood of the successful wearing of contact lenses were the absence of suppression and the presence of constant diplopia. The testing was done with the use of an afocal lens which eliminated aniseikonia. Eyes satisfactorily fitted will be restored to full binocular vision. Beulah Cushman.

Riggs, L. A., Armington, J. C., and Ratliff, F. **Motions of the retinal image during fixation.** J. Optic. Soc. America **44**: 315-321, April, 1954.

This further study of ocular motion showed that during an image exposure of 0.1 second, the retinal image moved 25 seconds of arc, which is the diameter of a retinal cone. Therefore, such an exposure will stop the motion of a retinal image as in a photographic snapshot. Longer exposures than 0.1 second would be too coarse for many visual tests.

Paul W. Miles.

Sachsenweger, R. **Anomalies of refraction and their influence upon depth perception.** Arch. f. Ophth. **154**:617-636, 1954.

The author studied the depth perception of more than 1,000 subjects 10 to 70 years of age. He used the three rods device at a distance of 5 meters. The diameters of the rods were varied according to the visual acuity of the examinees. The errors in depth perception varied with the occupation of the subject. Farmers showed 4.5 cm. deviation for 5 m. distance, mechanics 2 cm., physicians 2.1 cm., younger students 1.8 cm. Errors were smaller when the distance of the middle rod was increased, they became larger when the distance was decreased. The author explains this observation with the help of the Hering-Hillebrand-horopter which causes distant objects arranged in a fronto-parallel line to appear in a line having a concavity directed towards the observer. In moderate anisometropias the depth perceptions were found to be somewhat diminished, in higher degrees of anisometropia stereoscopic vision became suppressed. (9 figures, 46 references)

Ernst Schmerl.

Shapland, C. Dee. **Changes in the refraction of the eye following the operation of lamellar scleral resection.** Tr. Ophth. Soc. U. Kingdom **73**:205-238, 1953.

The author discusses lamellar resection in cases of retinal detachment and shows that the operation could be employed theoretically only to correct or diminish a higher degree of regular astigmatism by placing the sclerectomy at right angles to the meridian of the natural lesser curvature of the cornea or to reduce a high degree of myopia in the absence of a retinal detachment. The positive value of the operation in treatment of retinal detachment seems now to have been proved. (15 figures, 6 tables)

Beulah Cushman.

Strampelli, B. **The tolerance of the eye to acrylic lenses in the anterior chamber in aphakia and in refractive errors.** Ann. di ottal. e clin. ocul. **80**:75-82, 1954.

The author has constructed special acrylic lenses which can be implanted into the anterior chamber and avoid thus some of the difficulties experienced in the use of Ridley's acrylic lens implants. The implants have a rectangular shape, about 0.5 × 4 × 11 mm., one end of the rectangle ending in an arrow-shaped point; the other end has a central incision which gives it the form of the end of an arrow. The central part is occupied by a meniscus lens (convex or concave) with a radius of curvature of 12 mm. and a diameter of 6 mm. These lenses are implanted into the anterior chamber through a 6-mm. wide incision on the limbus. The three points at the extreme ends of the implant rest in the iridocorneal angle, allowing a firm seat with a minimum of actual contact.

The implants were used in two cases of aphakia and one of extreme myopia (−24D). They were tolerated from the first day without any reaction. The final vision is not reported. In the case of myopia the degree of nearsightedness was reduced to −4D and vision was "markedly improved." In the latter case a peripheral iridectomy was done in order to avoid a disturbance of the circulation of the aqueous. The period of observation in all cases was two to three months.

John J. Stern.

Walls, Gordon L. **The filling-in process.** Am. J. Optometry **31**:329-341, July, 1954.

In this well written paper, a new theory of visual perception is developed. Because of rapid loss of stimulus value of any constant illumination of the retina, it is only the contour line or border which maintains the cerebral image. This can exist only because micronystagmus keeps the contour line stimulating "off" and "on" receptors repeatedly. Whether or not receptors within the homogeneous interior of an image are intact and functional (blind spot, disease of retinal or cerebral transmission) the interior "fills in," and appears real. This would explain the failure of the subjective methods of testing visual fields by looking for defects in a 30° grid pattern. Paul W. Miles.

Weale, R. A. **Retinal irradiation and aniseikonia.** Brit. J. Ophth. **38**:248-249, April, 1954.

In the Pulfrich effect, a stereo-phenomenon, the pendulum bob appears to make rotatory excursions or ellipses. The effects of varying the illumination on same-sized objects, filters and slits were studied. Filters can reduce the apparent size owing to the reduction of retinal irradiation. Distortion of space resulted from a reduction of apparent size of targets. The flattening of the ellipse is due to variations in the latent period of vision.

Orwyn H. Ellis.

5

DIAGNOSIS AND THERAPY

Agarwal, L. P., and Gupta, B. M. L. **Sulphonamides and antibiotic drugs in the treatment of trachoma.** Brit. J. Ophth. **38**:119-122, Feb., 1954.

In order to evaluate treatment with the recommended antibiotics the authors studied terramycin, chloramphenicol, aureomycin and sulphonamides given both locally and orally under well-controlled conditions. Patients who showed inclusion bodies in slides from conjunctival scraping were selected. To a certain extent all the drugs were effective; treatment with terramycin was the least effective and with sulfonamide products the most. Orwyn H. Ellis.

Craig, P. C. **Sterile air in intraocular surgery.** A.M.A. Arch. Ophth. **51**:701, May, 1954.

Air is drawn repeatedly into a syringe through sterile cotton and tubing to provide a sterial media for injections of air into the anterior chamber. G. S. Tyner.

Cruickshank, R., Sorsby, A., Ainsli, D., and Saubermann, G. **Symposium. The scope of antibiotics and chemotherapeutic agents in ophthalmology.** Tr. Ophth. Soc. U. Kingdom **73**:153-194, 1953.

Cruickshank, Robert. **Some general considerations in the use of antibiotics.** pp. 153-159.

The first principle in the use of antibiotics is to attempt to treat specific infections, not clinical syndromes. Collaboration between the clinician and the bacteriologist is needed to identify the organisms and to determine the sensitivity to the antibiotics. The action of the drug may be either bactericidal or bacteriostatic. All these drugs exert their action on actively multiplying organisms; their concentration at the site of infection should be two to four times as great as the bactericidal or inhibitory concentration. The aim should be to give enough of the drug to restrain the invader until the host tissue is able to overcome the infection. Tissue response in overcoming infection and establishing resistance or immunity is an important factor; it is at its best in childhood and adult life and poorest in infancy and old age.

Toxicity may be manifested as local irritative or necrotic reactions, as allergic reactions, or as secondary effects from the replacement of flora, vitamin lack following elimination of the normal flora, or as specific toxic effects. Bacteria may be naturally resistant or acquire resistance to any particular antibiotic. Resistance may often be avoided by the administration of two similar drugs acting synergistically.

Sorsby, Arnold. **Experimental considerations in the use of antibiotics.** pp. 161-176.

Tentative experiments were carried out to increase permeability of the eye. The sulphonamides penetrate fairly readily into the aqueous while sulphadimidins, sulphadiazine and sulphamerazine give considerably higher levels than sulfathiazole. Penicillin levels in the aqueous are fairly proportional to the systemic doses given. Still higher levels can be obtained by combining subconjunctival injection of penicillin with adrenalin. Streptomycin is toxic in high doses; 0.5 mg. is the highest dose that can be given locally. In general the sulphonamides and penicillin are effective against gram-positive bacteria, streptomycin is valuable against gram-negative bacteria, while the newer antibiotics such as aureomycin, chloramphenicol, and terramycin are effective against both gram-negative bacteria and some viruses. When using antibiotics, the correct diagnosis and adequate concentration are essential. Trachoma is under control and epidemic keratoconjunctivitis is amenable to treatment. (12 tables)

Ainslie, D. **Antibiotics and chemotherapeutic agents.** pp. 177-181.

Polymixin extracted from bacillus polymyxa was found to be a powerful antibiotic against gram-negative bacilli. Experimentally it was found to be water-soluble and effective in 0.25 ml. of 1,000,000 units/

ml. or less, and did not cause corneal edema. Of forty strains of Ps. pyocyanea tested, 70 percent were sensitive to 10 units/ml.

In experimental infections there was no substantial difference between polymixins B and E. The smallest subconjunctival dose of polymixin to prevent infection was about 50,000 units. Rabbits treated later in the course of infection also responded well. Clinically the results were satisfactory in the treatment of gram-negative bacillus infections. The results depend upon how early the diagnosis of the type of infection is made. The author has found that an ointment combined with aureomycin (effective against cocci) given at the same time is most effective, otherwise there is danger that secondarily infecting organisms will invade the cornea before it has time to heal.

Saubermann, G. **Experimental results with antibiotics for clinical use.** pp. 181-194.

The author exhibited experimental evidence to show that an infected eye can be saved with good function when treatment is started within 24 hours with the right antibiotic, chemotherapeutic agent or the right combination. Fulminating development often leaves no time to determine the organism or sensitivity, therefore a certain routine procedure must be followed. Prophylaxis or treatment in cases with an unknown organism is started with penicillin, the least toxic agent, combined with a sulphonamide. If the course of the inflammation is unchanged in a few hours the antibiotic is changed.

From experimental work by the author he showed the concentration of penicillin in human aqueous and vitreous after different forms of application; for penicillin the subconjunctival method was found to be the best. Aureomycin is best given intravenously and 2½ grams are administered in half an hour every six hours. Of streptomycin one gram is given subconjunctively and one gram intramuscularly. Iontophoresis was found to be impractical as was the use of corneal baths with aureomycin and streptomycin. The influence of antibiotic and chemotherapeutic agents was studied by the measurement of bacterial respiration with manometric analysis with the Barcroft-Warburg apparatus.

Beulah Cushman.

Eisenberg, I. J., Leopold, I. H., and Sklaroff, D. **Use of radioactive phosphorus in detection of intraocular neoplasms.** A.M.A. Arch. Ophth. **51**:633-641, May, 1954.

Radioactive phosphorus approaches the ideal isotope for detection of intraocular neoplasms; the safety factor is adequate and there is a detectable difference between P^{32} in normal and neoplastic tissue. An analysis of P^{32} readings in 49 cases is presented. 500 mc of P^{32} was injected intravenously. A one minute count was taken over each quadrant of the normal eye. The average count per minute at the site of the lesion was determined. By this method it was found that variations in the normal eye may run as high as 28 percent although generally it is less than 20 percent. The authors believe that a variation of 30 percent or more should be considered significant. The test is most reliable when applied to neoplasms in the anterior segment of the globe. Active inflammatory lesions may give a false positive. The test should be used in conjunction with and not replace other diagnostic methods.

G. S. Tyner.

Giordano, A. **Retinal lipoid extracts in myopia.** Ann. di ottal. e clin. ocul. **80**:113-130, 1954.

Cellulin Retinale, a lipoid extract of animal retinas, was injected intracutaneously for ten days in 24 patients with high myopia and myopic fundus changes. In several patients satisfactory results were obtained, consisting of improvement of vis-

ual acuity with and without correction, improvement of the fundus lesions, and a sense of well-being. John J. Stern.

Kenney, Sheila. **Curare in ophthalmology.** Tr. Ophth. Soc. U. Kingdom **73**: 679-685, 1953.

The use of curare produces relaxed muscles and an immobile eye under light anesthesia with early return to consciousness. The disadvantage is the tendency to increased bleeding. This can be controlled with the proper position of the patient before starting the anesthetic. Curare with local anesthesia was found to be particularly beneficial. One strives to produce good palpebral and global akinesia, to preserve the ability of the patient to move the eye on command, and to effect respiration as little as possible. One gives 1 to 2 grains of luminal the night before and again two hours before the surgery. Atropine is given one hour before surgery to prevent the salivation which may occur with curare. If the ocular tension is raised, a retrobulbar injection of procaine solution is given. The injection of curare into a vein on the back of the hand is begun when the local anesthetic is given (1 mg. tubocurarine per fourteen pounds body weight). Injection is continued until ptosis is produced (usually after 3 mg.) and more is given as needed to aid the relaxation. The hand grip is a useful guide to dosage. It is important to wait three full minutes after the last injection to avoid any interruption due to untoward reactions occurring during operation.

Beulah Cushman.

MacDonald, Alexander E. **Topical cortisone, some problems.** Tr. Ophth. Soc. U. Kingdom **73**:267-270, 1953.

The author reports on the topical use of cortisone. Its early use, in excess of the normal body supply, after epithelium has been reformed, will limit vascular invasion of the cornea and give less scarring. The use of cortisone for maintenance, when the normal body supply is embarrassed, should be constantly evaluated. Its insulin-like use to supply a deficiency may be prolonged. When allergens cannot be avoided, its use gives relief without cure.

Beulah Cushman.

Mooney, Alan. **A preliminary report on the topical use of Neosone.** Tr. Ophth. Soc. U. Kingdom **73**:691-695, 1953.

The Upjohn Company supplied an ointment combining 15 mg. cortisone and 5 mg. neomycin in drachm tubes with which 100 patients with palpebral, corneal, and conjunctival inflammation were treated. Most forms of corneal ulceration improved more quickly than with other forms of treatment. Beulah Cushman.

Neubert, Frank R. **Vascular patterns in the eye. (A preliminary report on a photographic study.)** Tr. Ophth. Soc. U. Kingdom **73**:251-260, 1953.

The report is made on a photographic study of the vascular patterns in infection of the eye, to see whether the vessels behaved similarly in tuberculosis, diabetes, syphilis or in hyperpiesis and if the diagnosis could be influenced by the pattern which they made. These hopes were not substantiated. This study included the conjunctival anastomosis between the anterior ciliary vessels and the posterior conjunctival vessels, the limbal plexus, the perforating vessels, and the vessels in general and their association with systemic disturbances. (8 figures)

Beulah Cushman.

Pereyra, R. F. **Cortisone in ophthalmology.** Arch. oftal. Buenos Aires **29**:29-37, Jan., 1954.

After a survey of the current literature on the subject, a report is presented on the good results of topical and systemic cortisone administration in miscellaneous ocular conditions. Two observations intended

to demonstrate the appearance of untoward side effects of cortisone drops (slight elevation of the systolic and especially the diastolic blood pressure in a case of arterial hypertension; transient, ill-defined abdominal pain in a person who suffered from no gastroenteric disorder) must be cautiously evaluated. (21 references) A. Urrets-Zavalía, Jr.

Perkins, E. S. **A simple applanation tonometer.** Tr. Ophth. Soc. U. Kingdom **73**:261-266, 1953.

The principle of applanation tonometry is that the area of flattening of the cornea produced by the application of a known weight by way of a flat surface is directly proportional to the intraocular pressure. The author feels the main advantages of the instrument are its simplicity, the absence of moving parts and its light weight, which means that the actual intraocular pressure is altered very little. (3 figures, 2 tables) Beulah Cushman.

Rosso, S. **The present usefulness of vitamin A in ophthalmology.** Ann. di ottal. e clin. ocul. **80**:31-42, 1954.

The author provides a rambling discussion of the "ocular disturbances connected with the menstrual syndrome" (hordeolum, meibomitis, chalazion, phlyctenular conjunctivitis, myopia of puberty, constriction of the visual field, heterophoria, episcleritis, capillarospastic retinopathy and bullous keratitis); disturbances of the menopause (defective dark-adaptation, difficulty of opening the eyes after sleep, absence of the lacrimal reflex, palpebral edema, precocious presbyopia); and "thyrotoxic disturbances with hemeralopia." No vitamin A determinations or dark adaptation tests were performed. All patients were cured with Vitamin A.

John J. Stern.

Schreck, Eugen. **A new instrument using electronic tubes for the therapeutic use of direct current.** Arch. f. Ophth. **155**: 79-96, 1954.

A new kind of a pantostat and the clinical indications for the use of weak direct current are described. (2 figures, 27 references) Ernst Schmerl.

Shah, Mahmud Ali. **The corneo-scleral track suture.** Brit. J. Ophth. **38**:239-241, April, 1954.

A new technique of corneo-scleral track suture is presented. An unthreaded needle is passed through the corneo-scleral junction and left in place. A cut through the limbus is made down to the needle. The needle is removed and a suture is threaded through the holes. The section can be made either with cataract knife or keratome and scissors. The advantages of preplaced corneo-scleral sutures for perfect opposition are enumerated.

Orwin H. Ellis.

Stallard, H. B. **The corneo-scleral suture.** Brit. J. Ophth. **38**:232-238, April, 1954.

The author describes his corneo-scleral suture which is a modification of MacLean's preplaced suture. A limbus-based flap is cut and then a groove is made at the limbus. The sutures are placed in the 11- and 1-o'clock positions and are looped around lid clamps to keep them out of the way. The suture is arranged to separate the edges of the corneo-scleral wound and to facilitate the completion of the section with cataract knife or keratome and scissors. Two sutures are more effective than one in checking postoperative hyphema.

Orwyn H. Ellis.

Wheeler, J. R. **Anesthesia and akinesia in ocular surgery.** Tr. Ophth. Soc. U. Kingdom **73**:505-512, 1953.

The author believes that local and regional anesthesia is preferable to general where practicable and curare may be given to augment it in the hypersensitive pa-

tient. General anesthesia should be employed in non-cooperative patients, such as the very neurotic, the deaf or the very young. Beulah Cushman.

6

OCULAR MOTILITY

Anderson, J. Ringland. **Latent nystagmus and alternating hyperphoria.** Brit. J. Ophth. **38**:217-231, April, 1954.

Latent nystagmus is manifest with the occlusion of one eye, but not with both eyes open. The incidence of birth injury is high. The nystagmus is nearly always rhythmic, with the initial slow component towards the covered eye. In most cases an excessive action of one or both inferior oblique muscles was found, giving an alternating hyperphoria. The rotatory nystagmus is probably due to the imbalance of the oblique muscles. Many case reports are presented in detail.

Orwyn H. Ellis.

Lynn, Beatrice H. **Pitfalls in orthoptic treatment.** Tr. Ophth. Soc. U. Kingdom **73**:659-672, 1953.

The time to bring a child for advice is as soon as the squint is noticed. Better results have been obtained with binocular surgery in patients with a large angle of squint rather than a drastic operation in one eye. There is a definite tendency to reduce the amount of recession of the medial rectus and to increase the amount of resection of the lateral rectus.

Intermittent squints comprise one-quarter of all cases and are divided into 1. latent at all distances with glasses and manifest on removal of glasses, 2. latent with glasses for near and manifest for distance, and 3. latent with glasses for distance and manifest with glasses for near vision. The treatment of all three is a full correction of the refractive error, occlusion, and antisuppression exercises. Full correction in the early stages is beneficial but if worn too long may result in poor binocular visual acuity. Glasses must be reduced.

Beulah Cushman.

Raubitschek, E. **False torsion.** Ophthalmologica **127**:52-55, Jan., 1954. Roelofs, C. O. **The truth about false torsion.** Ophthalmologica **127**:56-61, Jan., 1954.

Both articles are responses to Pascal's paper on false torsion (cfr. Am. J. Ophth. 37:468, March, 1954), Raubitschek casting an affirmative and Roelofs a dissenting vote on Pascal's view that the retinal meridians during false torsion undergo a tilt opposite to that of the corresponding corneal meridians. The disagreement seems to be due to difficulties arising from the use of planar coordinates and could be resolved by the use of spherical coordinates as planes of references. On his large model of the human eyeball (cfr. Arch Ophth. 51:783, 1954), Quereau finds that during false torsion corresponding corneal and retinal meridians are tilted in the same direction with reference to the objective vertical plane. Both vertical meridians undergo (false) extorsion with reference to the objective vertical plane when the left eye, rotating around an axis in Listing's plane, looks up and to the left. (2 figures, 1 table, 3 references)

Peter C. Kronfeld.

Siebeck, Robert. **Disturbances of perception and awareness of disturbances in ocular paralyses.** Arch. f. Ophth. **155**:26-34, 1954.

The author studied the factors causing imaginary movements of objects in experimental and pathologic paralyses of the ocular muscles. In experiments on himself with curare and decamethonium he found that imaginary movements of objects occurred only when the head or the body was moving. Unsuccessful intention to move the eyes did not produce any imaginary movements of special objects or the

environment. With his ocular muscles experimentally paralyzed the author was aware of the impossibility to move his eyes. Similar observations were made in patients with ocular paralyses. When the normal eye was covered, imaginary movements occurred only with movements of the head or the body. From these observations and experiments, the author draws the conclusion that our impressions about the posture of our eyes is determined by the voluntary impulse (Hering, Kries) as well as by the achieved position of the eye. (35 references) Ernst Schmerl.

7

CONJUNCTIVA, CORNEA, SCLERA

Cristini, Giuseppe. **Fatty corneal dystrophy.** Rassegna ital. d'ottal. **23**:110-123, March-April, 1954.

Cristini reports a case of bilateral corneal dystrophy in a 48-year-old man. The diagnosis depended upon the biomicroscopic appearance of the cornea, the peripheral annular form of the lesion, its progressiveness, the absence of subjective symptoms and on its occurrence in uninjured eyes. Nonpenetrating, and later, total keratoplasty were performed, but were soon followed by continued loss of vision. The histopatholgy gives a clue to the diagnosis. These findings were lymphoplasma cells and eosinophile cells with an infiltrating lipoid degeneration and they suggest that the corneal disease is a monosymptomatic form of Hand-Schueller-Christian disease. (3 figures, 42 references) Eugene M. Blake.

De Paula, Xavier J. **Purulent ophthalmia.** Arq. brasil. de oftal. **16**:203-208, 1953.

The literature before 1942 is replete with opinions about ophthalmia neonatorum, in which little optimism is expressed. Many factors are to be considered in evaluating the prognosis, such as the virulence of the organism, corneal involvement, duration before treatment is begun and duration of contact of the organism with the ocular structures. The advent of penicillin has changed the prognosis drastically. The disease may now be considered curable. A case history is presented of a two-day-old infant with purulent conjunctivitis due to the gonococcus. Treatment consisted of removal of the pus and frequent instillations of penicillin solution. Dramatic improvement followed, with disappearance of the organisms from the discharge. An uncomplicated recovery resulted.

James W. Brennan.

Doggart, J. H. **Corneal calcification.** Tr. Ophth. Soc. U. Kingdom **73**:111-115, 1953.

The author reviews the knowledge of the calcium metabolism of the body. He looks forward to the time when the deposit of calcium in the corneal tissues may be better understood and prevented.

Beulah Cushman.

Guimaraes, Wilson **Keratoconus posticus circumscriptus.** Arq. brasil. de oftal. **16**:209-211, 1953.

In keratoconus posticus the posterior corneal surface has a curvature that is greater than normal and greater than the curvature of the anterior surface. The axial region is thinner than the periphery. Only about six cases have been reported. The anomaly is found in two types. In one, the entire posterior corneal surface is involved with a general thinness of the cornea. The condition remains stationary, appears in youth, and seems to be a developmental defect. The second type is more localized, and apparently is acquired, possibly as a result of trauma, as there is an associated corneal opacity.

A case history is presented of a 53-year-old man who had one eye enucleated in childhood. Vision in the remaining eye had always been poor, but had failed more

in recent years. The cause of poor vision had been attributed to a corneal leukoma. Visual acuity was 0.3 with the best correction. Examination revealed a disc-like formation in the cornea which was transparent, but surrounded by an opaque areola. Biomicroscopic examination showed thinness of the corneal substance in the affected region, with an increased curvature of the posterior corneal face. No history of trauma to the eye was elicited. No follow-up examinations were possible. (4 references) James W. Brennan.

King, E. F. **Keratoconus following thyroidectomy.** Tr. Ophth. Soc. U. Kingdom **73**:31-39, 1953.

The author discusses the question whether disturbed thyroid function is a significant factor in the pathogenesis of keratoconus in two patients. A woman, 47 years of age, had a thyroidectomy for thyrotoxicosis at the age of 25 years. Her health improved and the proptosis receded. Her condition remained satisfactory until the age of 39 years, then vision began to deteriorate and within three years bilateral keratoconus was found by retinoscopy and with the Placido disc. The apices were slightly down and out from the center of the cornea. No evidence of keratoconus was found with the slit-lamp. Contact lenses were fitted and the vision improved to 6/24 and 6/36 respectively.

In the second patient, a woman aged 27 years, in whom a thyroidectomy was done for thyrotoxicosis, a proptosis appeared soon afterwards and the vision gradually deteriorated. She was fitted with contact lenses and the vision improved to 6/9 and 6/12. (6 figures) Beulah Cushman.

Paternostro, V. **Paramino-benzoic acid in the treatment of the corneal manifestations of herpetic infections.** Ann. di ottal. e clin. ocul. **80**:175-180, 1954.

Paramino-benzoic acid by mouth (6 gm. daily) and intravenously (2 gm. daily) resulted in objective and subjective improvement in 8 of 12 cases of herpetic infection of the eye (four cases of disseminated herpetic keratitis, two of dendritic keratitis, one of superficial punctate keratitis and one of corneal ulcer).

John J. Stern.

Rouher, F. **Hydrocortone and atropine eczema.** Bull. Soc. d'ophtal. France **2**:200-204, Feb., 1954.

The author recommends hydrocortone in atropine-hypersensitivity conjunctivitis and dermatitis. Hydrocortone was so effective that occasionally the use of the offending agents did not have to be stopped. Thirteen relevant case histories were analyzed. (18 references)

Alice R. Deutsch.

Sapuppo, Concetto. **Keratoconus and glaucoma.** Gior. ital. oftal. **7**:64-74, Jan.-Feb., 1954.

In a case of bilateral keratoconus with an associated chronic glaucoma which was originally controlled in one eye by perforating diathermy-coagulation, the tension increased after treatment of the ectatic cornea by diathermy, necessitating a trephine operation. After a review of the possible causes of keratoconus, and a reference to the similar cases described by Alajmo and Simonelli, the author advises that before attempting surgical treatment of keratoconus one must investigate thoroughly to discover whether there is a tendency to glaucoma. The association of keratoconus and glaucoma is rare, but when present, treatment of the former condition may aggravate the latter. (2 figures, 29 references) V. Tabone.

Sivasubramaniam, Hoole T. **Hernia of the choroid treated by scleral grafting followed by direct suturing of the sclera.** Brit. J. Ophth. **38**:126-127, Feb., 1954.

A hernia of the choroid was treated

originally by excising the covering sclera and suturing in place a donor piece of sclera. A few days postoperatively it was seen that the hernia had recurred, that the scleral graft was attached at one side only but was adherent to the presenting choroid. At a second operation, after sutures were pre-placed, the scleral defect was closed directly over the choroid and scleral graft. Healing was uneventful with resulting good vision. The use of caps over scleral grafts like plastic corneal caps is suggested. Orwyn H. Ellis.

Weisser, C. W. **Fibrin film treatment of corneal diseases.** A.M.A. Arch. Ophth. **51**: 681-686, May, 1954.

Thromboplastin (thrombin U.S.P., 5,000 units and human plasma U.S.P. 2.5 cc), when applied properly, promotes healing of the corneal epithelium and relieves pain. The author cites its successful use in chemical burns, abrasions and corneal ulcers. G. S. Tyner.

8

UVEA, SYMPATHETIC DISEASE, AQUEOUS

Anastasi, G., and Bellavia, M. **Some abiotrophic alterations of the fundi.** Gior. ital. oftal. **7**:11-43, Jan.-Feb., 1954.

Familial choroidoretinal lesions are extensively reviewed. Four cases of familial macular degeneration and 12 of abiotrophic choroidal sclerosis are described. The authors conclude 1. that all forms of familial macular degeneration are different manifestations of abiotrophy of the neuro-epithelium and 2. that trauma and infections can precipitate abiotrophic lesions which start from the beginning in the neuro-epithelium. They suggest that choroidal sclerosis is essentially a degeneration of the choroid and not an arteriosclerotic lesion and that it is distinguished from macular degeneration by this fact and by the clinical course. It probably forms part of the larger group of abiotrophy of the mesoderm. Tissue therapy is more efficacious in choroidal sclerosis than in familial macular degeneration. (19 figures, 73 references) V. Tabone.

Bartholome, Aguilar. **Nonperforating retrociliary diathermy in the treatment of hypertensive uveitis.** Arch. Soc. oftal. hispano-am. **13**:1315-1344, Nov., 1953.

A review of the literature on the treatment of hypertensive uveitis, and the technique, indications and results of nonperforating retrociliary diathermy coagulation with the pyrometric electrode is followed by a tabulated analysis and brief histories of the author's own seven cases. Two cases were subacute, three chronic, and four had seclusion of the pupil. The operation is simple and harmless, and may be repeated, or used after other unsuccessful tension-reducing operations. The ocular tension was reduced in 58 percent of his cases. It is effective in all cases of acute hypertension, when other surgical procedures are fraught with danger. Good results are obtained in chronic cases. When there is seclusion of the pupil it is totally ineffective. Atropine may be used postoperatively, and the effect of the operation may be augmented by a retrobulbar alcohol injection and paracentesis of the anterior chamber. The operation has but little effect on the visual acuity. It is compatible with all therapeutic procedures, and postoperative changes in refraction or corneal disturbances are transitory. (20 figures, 12 references)
Ray K. Daily.

Hobbs, H. E., and Simpson, E. D. L. **Systemic hormone treatment of posterior uveitis.** Tr. Ophth. Soc. U. Kingdom **73**: 531-546, 1953.

The authors examined the case histories of a group of patients who were blind from uveitis to determine if a particular etiologic factor seemed demonstrable

in the worst cases and to find the anatomic complications which led to blindness. Ten percent of patients certified were blind from some form of uveitis. Three of each ten patients were known to have been syphilitic, in the remaining seven no clear etiology was established although most of them had received tuberculin at some time. The anatomical factors were complicated cataracts in five and in two cases blindness resulted from secondary glaucoma. Vitreous abnormality in 18 cases of uveitis was of prime importance, and its resistance to the usual treatment led to the systemic treatment with ACTH. In general the clearing of the vitreous was accompanied by visual improvement except in one or two there was a macular lesion and visual improvement was less. A total of 27 patients was treated. The intramuscular route was found preferable and dosage of 20 mg. three times daily was used and occasionally increased to 40 mg. after three days in the absence of any undesirable effects. Treatment was given for a week or so, then gradually was discontinued by reducing the dose and frequency or by substituting cortisone tablets. Cortisone was given in 100-mg. doses daily for eight days, then gradually stopped by diminishing doses given orally. Patients with serious cardiovascular, renal, pulmonary disease or with diabetes mellitus and previous mental disorders were excluded from treatment with ACTH and cortisone. During treatment measures to determine and control fluid retention were used and potassium citrate was prescribed as a routine. All of the treatment was guided by associated internists. Beulah Cushman.

Koenig, I. J. **Malignant melanoma of the iris and ciliary body of a one-eyed patient.** A.M.A. Arch. Ophth. **51**:656-662, May, 1954.

In this most interesting and valuable case report a patient is described who had lived for an estimated 52 years with a melanoma of the iris and ciliary body which at autopsy was found to be malignant. X-ray treatments had been given over a long period. A successful cataract extraction had been performed without operative or post-operative complications. The patient obtained 6/6 vision post-operatively. She died of myocardial infarction. G. S. Tyner.

MacRae, A. **Prognosis in malignant melanoma of choroid and ciliary body. (President's address)** Tr. Ophth. Soc. U. Kingdom **73**:3-30, 1953.

The authort presents a statistical study of 66 patients with malignant melanoma of the uvea who were seen between 1927 and 1947. The after-history up to the middle of 1952 is included. In five patients there is doubt as to whether death should be ascribed to metastasis. The right eye was affected in 36 patients. The youngest patient was 23 years of age and the oldest 74, the average age in 61 cases was 52.7 years. There were 27 men and 34 women. Metastasis occurred as long as 25 years after enucleation. One patient only showed recurrence in the socket; the orbit was exenterated and the patient remained alive 13 years after this operation.

Twenty-five of the pathologic specimens were tested for reticulum content; in these cases the amount of pigment was related to prognosis, and the relation the size of the tumor was to the percent of recurrence and metastasis was evaluated. (6 tables, 7 figures) Beulah Cushman.

9

GLAUCOMA AND OCULAR TENSION

Carramazza, Filippo. **Pathologic and therapeutic review of the glaucomatous syndrome.** Rassegna ital. d'ottal. **23**:81-100, March-April, 1954.

This article is the introductory lecture presented to the faculty, students and

others at the time of Dr. Caramazza's elevation to the professorship of ophthalmology at Bologna University. It is an excellent review and summary of our knowledge of glaucoma. Eugene M. Blake.

Guenther, G., and van Beuningen, E. G. A. **The regulation of the ocular tension following iridencleisis.** Arch. f. Ophth. **155**:1-10, 1954.

More than 150 patients were followed for two to three years. The best site of the filtering area seems to between Schlemm's canal and Schwalbe's ring. The tension was found to be normalized in 87 percent of all patients with primary and secondary glaucoma. (1 figure, 5 tables, 9 references) Ernst Schmerl.

Hakim, A. E. Sohrab, **Argemone oil, sanguinarine, and epidemic-dropsy glaucoma.** Brit. J. Ophth. **38**:193-215, April, 1954.

This is a long, detailed description of the epidemiology, chemistry, toxicology, pharmacology and biochemistry of the plants concerned. Large numbers of rats were poisoned and the histopathology studied. Three fundamental actions were found: 1. anti-adrenalin, 2. histopathological action on cornea, filtration angle, retina and skin, specially in old rats, and 3. disturbance of fluid-tissue balance, which occurs particularly in protein depletion. A constrictive influence on outflow channels of the excised eye was recorded. Subconjunctival and anterior chamber injections produced an acute rise in ocular tension. The pathologic changes are similar to those of primary glaucoma. Adrenalin, ephedrine and BAL antagonize the tension-raising effect. It is suggested that injection of minute amounts of argemone as generalized contaminants of food might cause widespread glaucoma, since this is the only known substance capable of causing human glaucoma.

Orwyn H. Ellis.

Higgitt, A. C. **The dark-room test.** Brit. J. Ophth. **38**:242-247, April, 1954.

In the dark-room test a small rise of tension was present in normal eyes and eyes with chronic simple glaucoma. In congestive glaucoma 78 percent of cases showed a positive result. The cause of the increased tension appeared to be obstruction of the angle of the anterior chamber by the root of the iris which was visible with the slitlamp. The positive response to the test was not altered by surgery on a number of eyes. Extensive anterior synechiae may alter the angle so that it may be closed by some mechanism other than peripheral iris bombé.

Orwyn H. Ellis.

Horwich, H., and Breinin, G. M. **Phasic variations in tonography.** A.M.A. Arch. Ophth. **51**:687-694, May, 1954.

Twenty patients with proven open-angle glaucoma were admitted to a hospital and subjected to tonography after miotics had been discontinued. Readings were taken at 7 A.M., 12 noon, 5 P.M., and 10 P.M. in order to study the diurnal variation in ocular tension. The authors found that the rising phase of tension was accompanied by decreased facility of aqueous flow. Tonographs were negative on proven cases of glaucoma during certain hours of the day as a result of the phasic variation in ocular tension.

G. S. Tyner.

Kalt, M., Decaudin, A., and Loisillier, F. **Torpid iritis following antiglaucomatous operations.** Bull. Soc. d'ophtal. France **2**:193-194, Feb., 1954.

The authors contribute a survey on the often deleterious iritis following antiglaucomatous surgery, especially Elliot's trephining operation. Early postoperative use of cortone and perferrably hydrocortone was recommended.

Alice R. Deutsch.

Suda, K., Inamochi, J., and Ikuta, M. **Local regulation of ocular tension in two types of primary glaucoma, one controlled by miotics, another so-called low-tension glaucoma.** Acta. Soc. Ophth. Japan **58**: 633-636, July, 1954.

The lowering of the ocular tension after a compression of the eyeball with 50 gm. for ten minutes, was 75 and 30 percent on the average in normal and decompensated glaucomatous eyes respectively. In 17 cases of glaucoma compensated by miotics and in 16 cases of so-called low tension glaucoma, the lowering was 50 and 43 percent on the average respectively. After surgery, the lowering became 71 and 68 percent respectively in the last two groups. The authors consider that in glaucoma eyes there is still a disturbance in the regulation of the ocular tension even if the tension seems to be controlled by some means. (8 tables, 3 references)

Yukihiko Mitsui.

Suda, K., Oyama, T., and Miyata, N. **Diagnostic value of red visual field in early glaucoma, especially under low illumination.** Acta Soc. Ophth. Japan **58**: 642-647, July, 1954.

Seven eyes of early glaucoma with normal peripheral field for white were selected. The peripheral fields in these cases and some other control cases were determined by white and red test-objects under a high (2,000 lux) and a low (20 lux) illumination. A concentric contraction of the red field under the low illumination seemed to be characteristic for glaucoma and of diagnostic value. Seidel-Bjerrum's scotoma was demonstrated only in two of the seven glaucomatous eyes. The authors conclude that a determination of the red field under 20 lux illumination is of great value in the diagnosis of early glaucoma. (5 tables, 12 references)

Yukihiko Mitsui.

10

CRYSTALLINE LENS

Rouher, F. **The use of a solution of neosinephrine, and an anesthetic in the preparation for cataract operations.** Bull. Soc. d'ophthal. France **2**:198-200, Feb., 1954.

Rouher suggests the use of a mixture of 5-percent neosinephrine and 2-percent pontocaine in drop form, 20 and 10 minutes before cataract operations. He had favorable results in about 66 percent of his patients. René Nectoux achieved a satisfactory dilatation of the pupils in 90 percent of patients by using neosinephrine 60 and 30 minutes before surgery. Nevertheless, he considers the American manufactured 10 percent neosinephrine a safe and very reliable mydriatic.

Alice R. Deutsch.

Williamson-Noble, F. A. **Cataract extraction after a glaucoma operation.** Tr. Ophth. Soc. U. Kingdom **73**:585-595, 1953.

The author feels that cataract is more apt to develop after a glaucoma operation when there has been a delay in the reformation of the anterior chamber. He prefers iridencleisis to trephining because the anterior chamber is nearly always refilled at the time of the first dressing. Conventional cataract incision with a Graefe knife can easily be done through a trephine bleb. The latter will become reestablished later. After iridencleisis the filtration channel is narrower and more easily blocked by the healing of a cataract incision. Iris tissue in the wound increases the risk of sympathetic ophthalmitis. Removal of the lens from the lower conjunctival cul-de-sac may be done, but is more difficult.

The author describes a method of placing sutures over the cornea and anchoring them in the insertion of the rectus muscles. To protect the cornea he puts a disc of egg membrane or transparent flexible plastic under the sutures.

Couching may be of advantage in an only eye with glaucoma. Beulah Cushman.

11
RETINA AND VITREOUS

Aberastain, T. G. **Unilateral retinitis pigmentosa.** Arch. oftal. Buenos Aires **29**: 38-40, Jan., 1954.

After some doctrinal remarks which have been drawn freely from Franceschetti and Klein's classical monograph on the subject (cf. Franceschetti, A., and Klein, D.: The tapeto-retinal degenerations, in Franceschetti, A., et al.: Clinical and Social Aspects of Heredity in Ophthalmology. XVI Conc. Ophth. Internat. Acta vol. 1, pp. 187-188, 1950), a case of unilateral retinitis pigmentosa is described. A woman, 28 years old, whose family history was noncontributory, had been noticing a progressive visual failure on the right for the past eight years. The exotropic right eye exhibited the typical ophthalmoscopic picture of retinitis pigmentosa; the left eye, on the contrary, was entirely normal. Vision was limited to light perception in the affected right eye, and was normal in the left. It is unfortunate that no precise data concerning the status of the visual fields are supplied and that no adaptometric studies were performed. These exremely rare cases can be ascribed to the partial, asymmetrical manifestation of a recessive gene in a heterozygous individual. (1 figure, 8 references) A. Urrets-Zavalia, Jr.

Balacco, F. **Nodular syphiloma of the retina.** Arch. f. Ophth. **154**:574-578, 1954.

A syphiloma is described which had developed in a syphilitic person after an injury by contusion. The fundus showed a major nodule near the foveal area. The diagnosis seemed to be confirmed ex juvantibus: mercury, bismuth and penicillin caused the nodule to disappear, leaving behind a chorioretinal scar. (1 figure) Ernst Schmerl.

Chinaglia, V., and Foroni, O. **The relations between the light sense, the melanophoric hormone (intermedine) and the diencephalic centers. The action of the melanophoric hormone.** Ann. di ottal. e clin. ocul. **80**: 1-24, 1954.

Intermedine, the melanophoric hormone of the hypophysis, was used in therapeutic trials as drops or subconjunctival injections in 31 patients with pigment degeneration of the retina, myopia, albinism and essential hemeralopia. In five of seven cases of tapeto-retinal degeneration some improvement (1/10 to 3/10) of the visual acuity and of the light sense was obtained. In 17 of 22 cases of myopia an even better result was observed (1/10 to 4/10). The fields and the changes in the fundus remained unaltered in all cases. The effect of the hormone is interpreted as a "diencephalo-hypophyseal reflex action in accord with the newer physioanatomical and neurobiological knowledge."

John J. Stern.

Lester, A. and Zwink, F. B. **The course of thrombosis of the retinal veins.** Tr. Ophth. Soc. U. Kingdom **73**:55-71, 1953.

Stimulated by experience with a patient who recovered good vision in five months after thrombosis of the central vein, the authors studied other patients and were led to this investigation. The variety of anatomical end-results was especially noticeable. In the first group there was minimal new vessel formation. The second group showed efficient-looking venous by-passing at the optic disc. In the third group the permanent damage was greater than in the preceding groups and the main feature was the pattern of gross vascular changes. The last group showed gross vitreous change. The degree of visual recovery depended largely on the extent of damage to the macula, and the more extensive the vascular changes the greater the chances of macular damage. The final vision is approximately the same

as at the outset, with exceptions that some recover well after a bad beginning while the others show progressive deterioration. Treatment most commonly used was the giving of anticoagulants and its value was based on the reduction of the incidence of glaucoma in preventing anatomical and functional deterioration, and in improving visual acuity. An objection to the use of anti-coagulants is the risk of causing hemorrhage in the kidneys, alimentary canal, or elsewhere, or exciting fresh hemorrhage in the eye. One other method of treatment, the intravenous injection of novocaine, is based on the theory that occlusion is due to spasm of the accompanying artery and consequent ischemia of the retina. The novocaine causes arterial dilation by inhibition of the autonomic nervous system.

Beulah Cushman.

Pasternostro, V. **Pseudotumor of the retina (clinical observation and general considerations).** Ann. di ottal. e clin. ocul. **80**:181, 1954.

An irregular mass about 3 mm. in diameter and about 2 to 3½ diopters high in the posterior pole is described. The mass was white with yellow reflexes, spongy, with sharp edges, and surrounded the superior temporal artery. No change was observed for three years. No diagnosis of the character of the tumor was arrived at.

John J. Stern.

Stallard, H. B. **Annular peripheral retinal detachment.** Brit. J. Ophth. **38**:115-118, Feb., 1954.

The author presents six cases of bilaternal annular peripheral retinal detachment. The condition is most important because of the differential diagnosis between this condition and ring sarcoma of the choroid. Three cases were operated on successfully with nonpenetrating and penetrating diathermy.

Orwyn H. Ellis.

Wagener, H. P. **Diseases of the retina and optic nerve.** A.M.A. Arch. Ophth. **51**: 703-724, May, 1954.

The pertinent literature for the year is abstracted and reviewed. G. S. Tyner.

12

OPTIC NERVE AND CHIASM

Klauder, J. V., and Gross, B. A. **Results of penicillin, cortisone and nonpenicillin treatment of syphilitic optic atrophy, with report of clinical observations.** Am. J. Syph. **38**:270-287, July, 1954.

The authors treated 104 patients with syphilitic optic atrophy. Of these, five were congenital, the remainder acquired. Findings were checked against 81 untreated patients, and 86 pre-penicillin treatment results. Three schedules were used: 39 patients were given 4.2 million units in 7½ days; 29 were given 6 million units in 7½ days; and 36 were given 11 million units in 14 days. In some cases this therapy was supplemented with fever and metal chemotherapy, and more recently with cortisone. The results were checked by visual fields and visual acuity tests every three months for long periods. All three schedules produced about the same results; approximately 25 percent of each group become blind. Of the unfavorable results it was noted that some were "rapid progressors" and some "slow progressors." Cortisone appeared promising in the latter group, but is not advised as routine therapy. If the patient is not blind within three years after the onset, then the prognosis is good.

Harry Horwich.

Lyle, T. Keith. **Some pitfalls in the diagnosis of plerocephalic oedema.** Tr. Ophth. Soc. U. Kingdom **73**:87-102, 1953.

Plerocephalic edema is a term introduced by Traquair in 1946 to indicate edema of the disc due to raised intracranial pressure. The earliest signs are 1.

reddish discoloration of the disc, 2. blurring of the disc edges and gray streaks due to edema along the vessels (the blurring usually begins at the upper and lower poles of the disc, then spreads to its nasal margin, finally involving the temporal margin), and 3. engorgement of the veins. The ratio in caliber between veins and arteries increases from 3:2 to 4:2 or even 5:2. Plerocephalic edema must be differentiated from the edema of the optic disc which occurs as part of general retinal edema in hypertensive retinopathy, renal retinopathy, thrombosis of the central vein, or occlusion of the central artery. It must also be differentiated from edema of optic disc which is part of a localized retinal edema due to focal retinal lesions such as juxta-neural choroiditis of Jensen or posterior uveitis, from pseudo-neuritis, a congenital anomaly of the optic disc, and from opaque nerve fibers surrounding the optic disc.

Beulah Cushman.

13

NEURO-OPHTHALMOLOGY

Carreras Duran, Buenaventura. **The subjective symptomatology of annular scotoma.** Arch. Soc. oftal. hispano-am. **13**: 1241-1248, Nov., 1953.

The author reports the case of a patient who had an annular scotoma which left uninvolved an area of 8 to 10 degrees around the fixation point, and about 20 degrees at the periphery. The patient complained of poor vision at the intermediate distance. He could see near and far away, but everything became blurred as people advanced toward him. The eyes were externally normal and the media and fundi were normal except for a moderate vascular hypertension. The examination of the visual fields revealed an annular scotoma 15 to 20 degrees wide around the fixation point, leaving free a central area 5 degrees in diameter. It was evident that the free macula permitted the patient to have clear vision for near; but as the image of objects fell on the diseased area of the retina her vision became blurred. The patient had a general hypertension, with a blood pressure of 185/110. After a review of the etiology of annular scotomata the author concludes that its pathogenesis in this patient was on a vascular basis, probably caused by compression of the borders of the chiasm or optic nerves by calcified internal carotid arteries. The patient died five months later from apoplexy. (2 figures)

Ray K. Daily.

Chamlin, M. and Davidoff, L. M. **Homonymous hemianopia in multiple sclerosis.** Neurology **4**:429-437, June, 1954.

The authors describe in detail four cases of homonymous hemianopia in multiple sclerosis. Their cases were diagnosed as multiple sclerosis clinically. An accompanying defect noted was a paracentral scotoma. They comment on how extremely rare homonymous hemianopia is in multiple sclerosis, and suggest that possibly this is because the posterior visual pathways are involved so much later in the disease than the anterior portion.

Harry Horwich.

Jefferson, Geoffrey. **Bowman Lecture. Concerning injuries, aneurysms and tumors involving the cavernous sinus.** Tr. Ophth. Soc. U. Kingdom **73**:117-151, 1953.

There is a tendency in teaching to exaggerate the venous part of the sinus perhaps because the cavernous sinus contains the very important internal arotid artery and five nerve trunks. The proptosis in cavernous septic thrombosis is due to thrombosis of the orbital veins and cellulitis in the orbital tissues rather than to back pressure in the sinus. Trauma to the cavernous sinus usually injures more than one of the oculomotor nerves and perhaps the trigeminal as well. The motor nerves

suffer much more than the trigeminal divisions. A frequent diagnostic manifestation is the bruit. It should be looked for in all injuries to the head. The syndrome of the orbital apex includes injury to the optic nerve in the orbit as well as at its junction with the sphenoidal fissure.

Carotico-cavernous aneurysms have been made familiar by Sattler, Dandy and others. It is difficult to decide whether the injury occurs at the moment of injury or during the subsequent days. The bruit may be heard during the first day or in from one to five weeks, no one can know when they actually begin. Arterio-venous aneurysms can form and burst into the carvernous sinus. Ocular palsies are more often due to disturbances in the soft tissue of the orbit. The variable diplopias in which the muscles are at fault are usually due to orbital causes. Saccular aneurysms of the carotid lying in the cavenous sinus are more common than in that portion which is in the neck and may be the result of a developmental disturbance in the artery. These aneurysms are most commonly found in women of middle age or over. A clue to the situation of a saccular aneurysm is the extent of the numbness of the face. A lesion at the posterior end causes almost total numbness and ipsilateral paralysis of the masticatory muscles, of the middle portion numbness of forehead, eye and cheek, of the anterior end, numbness of forehead and eye only. (12 figures)

Beulah Cushman.

Leoz de Fuente, Gustavo. **Ocular symptomology of meningiomas at the base of the skull.** Arch. Soc. oftal hispano-am. **13**: 1249-1286, Nov., 1953.

This is a comprehensive discussion of the ocular symptoms of olfactory meningiomas, suprasellar mengiomas and meningiomas of the small wing of the sphenoid, as they were encountered in a nine-year period in the neurologic and psychiatric service of the Madrid General Hospital and the Institute of Neuro-Surgery. There was bilateral papilledema in six of 13 cases of olfactory meningioma, bilateral optic atrophy in three, papilledema, in one eye and secondary optic atrophy in the other eye of one patient, and a normal fundus in two. The visual fields were significant only in two cases. In one case with bilateral choked discs there was a loss of the left nasal field, and a constriction of the right inferior sector. In the second case, with optic atrophy there was left homonymous hemianopsia with constriction of the left inferior nasal quadrant. Unless surgery is done early, the prognosis for restoration of visual acuity in cases of olfactory meningiomas is poor. In 12 patients with suprasellar meningioma, in which ocular disturbances are frequently the first symptoms of the lesion, there were seven cases of primary optic atrophy, two cases of atypical Foster-Kennedy syndrome, one of bilateral choked disc, and in two the fundi were normal. Disturbances in visual acuity precede objective findings. In none of these cases did the author encounter the preatrophic papilla described by Cushing and Walker. The visual fields varied with the size and position of the tumor. Of ten patients with meningioma originating from the smaller wing of the sphenoid, eight had fundus changes, four had bilateral choked discs, two secondary optic atrophy, two the Foster-Kennedy syndrome, one an atypical Foster-Kennedy syndrome and one a normal fundus. A bilateral primary optic atrophy was not encountered in this group. One patient had a quadrant homonymous hemianopsia and one a marked binasal constriction. There was no exophthalmos in any of them. (24 figures, 25 references)

Ray K. Daily.

14

EYEBALL, ORBIT, SINUSES

Berke, R. N. **A modified Krönlein opera-**

tion. A.M.A. Arch. Ophth. **51**:609-632, May, 1954.

In this comprehensive discussion of the subject a simplified technique for the Krönlein operation is described in detail. This technique gives a greater exposure while leaving a less conspicuous scar. It results in no damage to the orbital or periorbital structures. The operation is indicated when the vision of the exopthalmic eye is threatened, when a primary retrobulbar malignant neoplasm is suspected, for cosmetic reasons, or for ligation of vessels in the retrobulbar space. It is contraindicated when the orbital tumor is anterior to the globe, when the retrobulbar space is invaded from adjacent structures, when the tumor is metastatic, when pseudotumor is present and when the exophthalmos is caused by certain systemic diseases such as thyroid disfunction, lymphosarcoma, Paget's disease, and others.

G. S. Tyner.

Chinaglia, V., and Santelli, F. **Cylindroma of the orbit.** Rassegna ital. d'ottal. **23**:124-153, March-April, 1954.

Various writers have stated that cylindromata of the orbit occur in percentages varying from five to 30. The author describes the case of a tumor of the lacrymal gland, which recurred two months after removal, in spite of X-ray therapy promptly given. The histologic examination of both the primary and the recurrent growth showed a mixed cylindroma, more clearly defined in the second growth. The author discusses mixed tumors and the diverse theories of the cellular changes fully. He believes that these tumors are essentially epithelial and that the cylindroma represents a metaplasia. (11 figures, 101 references) Eugene M. Blake.

Launay, C., Auvert, F., Verliac, F., and Grenier, B. **A case of toxoplasmosis with unilateral exophthalmus.** Bull. Soc. d'ophtal. France **2**:187-193, Feb., 1954.

The authors describe a six-weeks-old infant with typical cerebral and ocular signs of toxoplasmosis. The child also had considerable monocular exophthalmus which remained unchanged during an observation period of five months. The authors discussed the possibilities of a specific inflammation of the sheath of the optic nerve in toxoplasmosis. (5 figures)

Alice R. Deutsch.

Rodríguez Barrios, R., Arana Iñíguez, R., Medoc, J., and San Julián, J. **Orbital fibroma.** Arch. oftal. Buenos Aires **29**:43-54, Jan., 1954.

A most infrequent mesenchymal tumor of the orbit, fibroma, has been observed in young adults as a slowly growing hard mass, which eventually may become exceedingly large, is usually encapsulated and consists of collagenous tissue (i.e., of connective fibers, fibrocytes and blood vessels in variable proportion). Histologically it must be differentiated from the two main types of peripheral glioma: the neurinoma and the neurofibroma. Surgical removal is ordinarily easy and recurrences have not been recorded.

The case of a 45-year-old woman is presented, in which a progressive exophthalmos had been noticed on the right side for the last 15 years. On examination, a unilateral, marked proptosis was seen; although the globe was somewhat displaced downwards, there was no limitation of movements and binocular vision was intact. Exophthalmometric readings were as follows: O.D., 28 mm.; O.S., 18 mm. The visual acuity was 20/40 on the right side and 20/20 on the left. Fundus examination showed a slight papilledema in the affected eye. By disclosing an area of abnormal vascularization at the level of the internal two-thirds of the orbital pyramid, arteriography was of great help inasmuch as it permitted an accurate preoperative topical diagnosis. Extirpation was performed through a transcranial approach;

recovery was uneventful. Pathological examination established beyond any doubt the fibromatous character of the neoplasm. (21 references) A. Urrets-Zavalia, Jr.

15

EYELIDS, LACRIMAL APPARATUS

Paternostro, V. **So-called consensual blepharoptosis.** Ann. di ottal. e clin. ocul. **80**:167-174, 1954.

The case described occurred in a syphilitic patient. When the patient looked straight at a distant object and the left upper lid was pushed down with a finger, the right upper lid (of an amblyopic eye) oscillated slightly and then drooped completely. When the seeing left eye was covered with the hand, both the right and the left upper lids closed without a contraction of the orbicularis muscle. When the patient was now asked to open the eyes, he would merely accomplish a slight elevation of the lids by using the frontalis muscle. The pathogenesis of the lesion is discussed in detail. John J. Stern.

Paternostro, V. **Restitution of the patency of the lacrimal pathways by insertion of a plastic tube.** Ann. di ottal. e clin. ocul. **80**:161-166, 1954.

Ten patients with simple epiphora or dacryocystitis were treated. After anesthetizing the area with instillations and injections, Bowman probes Nos. 1 to 6 are introduced. The canaliculus is split right to the lacrimal sac, and a specially constructed tube of acrylic resin with ridges which prevent it from slipping is mounted on a Weber probe and inserted in the lacrimal duct. After four days the probe is removed from the tube and the implant tested by an irrigation. This irrigation is repeated daily for one week and combined with instillations of vasoconstrictors or antibiotics. The results were satisfactory in most cases. John J. Stern.

Rubinstein, Kazmierz. **Vaccinia of the eyelids.** Tr. Ophth. Soc. U. Kingdom **73**: 601-607, 1953.

The author describes an auto-inoculated patient with vaccinia of the caruncle, the lacrimal puncta, and the lower lid, with enlargement of the submandibular and preauricular glands. A drop of serum was removed and inoculated on the allantoic membranes of chick embryos in the virus laboratory. A typical growth was obtained after a few passages and a good membrane was photographed, sectioned and stained for virus. Healing took place rapidly after treatment with aureomycin systemically.
Beulah Cushman.

Schimek, R. A. **New techniques in ptosis surgery.** Henry Ford Hosp. Bull. **2**: 89-95, June, 1954.

The author states that operations upon the levator are the procedures of choice. He reviews Guyton and Iliff modifications of the Blaskovics technique for patients who have some levator function, and then suggests a modification of his own for ptosis with levator action. He splits the levator longitudinally, brings the two free ends up through tunnels in the upper lid and sutures them to the frontalis muscle. He considers this method useful even when others fail. (19 figures)
Harry Horwich.

Schofield, A. L. **A review of burns of the eyelids and their treatment.** Brit. J. Plastic Surg. **7**:67-91, April, 1954.

The surgical repair and late results are described for 144 patients with burns of the eyelids. 837 operations were performed on these lids by several surgeons, all of whom employed the epithelial outlay graft technique of Gillis, as modified by McIndoe in repairing the causalties of World War II. There are many photographs and a few drawings in the 21 figures which accompany the article. Intermarginal lid adhesions were not used, so larger grafts

were necessitated to prevent postoperative irregularities of the lid margin from contraction. (21 figures, 2 references)

Alston Callahan.

16
TUMORS

Bottino, C. **Neoplastic process of the lid of probable disontogenetic origin (cystic epithelioma).** Ann. di ottal. e clin. ocul. **80**:55-59, 1954.

A small nodule was observed on the lid margin of a man, 58 years of age. The skin over it was not mobile and showed some dyskeratotic desquamation. The nodule was excised in healthy tissue, without recurrence or metastasis for one year. The tumor was characterized histologically by a marked epithelial proliferation, delineated by cylindrical cells of the basal cell type and presenting some keratinization, pseudocystic elements consisting of cavities filled with cell detritus, and an absence of infiltration of the surrounding tissue. It was diagnosed as a basilioma.

John J. Stern.

Fontana, Giuseppe. **Symmetrical, pigmented epithelioma of the upper and lower eyelids in a young subject.** Rassegna ital. d'ottal. **23**:101-109, March-April, 1954.

The patient described had small blackish newgrowths upon the margin of the upper and lower eyelids of the left eye at birth. They were situated in symmetrical positions at the junction of the middle and outer third of the lids. The growths were conical, the size of a grain of wheat when first examined. They had increased to the size of a large pea by the time the child was three months of age, when the eye itself and the general condition appeared to be normal. At this time the masses were excised and the laboratory reported them to be melanotic, spindle-celled epitheliomata with characteristics of malignancy. During the next six months there was no evidence of recurrence or of metastasis. The cosmetic result was excellent.

The author gives a comprehensive review of the subject of malignant newgrowths in early life and concludes that tumors in babies are usually congenital and embryonic. Such tumors show characteristics of precosity and activity with many mitotic figures. (4 figures, 22 references)

Eugene M. Blake.

Lederman, M. **Radiotherapy of epibulbar malignant melanomata.** Tr. Ophth. Soc. U. Kingdom **73**:339-414, 1953.

The author reports his experience with 29 epibulbar malignant melanomas, 21 of which were treated by radiotherapy and eight were not. The main purpose of radiotherapy is to act as a complement to surgery in the attempt to preserve the patient's life and his eye. Radiotherapy is indicated chiefly when surgical treatment would entail loss of a good eye, when localized surgical measures have been incomplete, when the patient refuses operation, and for postoperative recurrences.

For limbal tumors, four localized exposures to 2,000 r. beta radiation, radio active phosphorous or radio active strontium are given over a period of four weeks. Tumors of the bulbar conjunctiva are treated in the same manner. A radon seed implant has been used for conjunctival melanosis and radio active tantalum mesh has also been used satisfactorily. Lid lesions were treated with low voltage X-rays in a dosage 6,000-7,000 r in four to six weeks. All epibulbar melanomata are radio resistant; small limbal tumors can be made to disappear by radiotherapy alone. Lesions of the bulbar conjunctiva are less radiosensitive and lid tumors are usually radio-insensitive. In selected cases of limbal melanomata radiotherapy can be successfully employed without preliminary excision. (5 tables, 8 figures)

Beulah Cushman.

Scuderi, G. **Metastatic carcinoma of the optic papilla and the choroid (clinical and histological study).** Ann. di ottal. e clin. ocul. **80**:143-160, 1954.

A case of metastatic dissemination in the optic nervehead and the choroid of a micro-cancer developing in a fibroadenoma of the breast is described. The ophthalmologic picture was characterized by papilledema which made the diagnosis difficult until new foci in the choroid clarified the issue.

John J. Stern.

Stallard, H. B. **Pathological study of retinoblastoma treated by radon seeds and radium disks.** A.M.A. Arch. Ophth. **51**: 573-588, May, 1954.

The undifferentiated cells of retinoblastoma are highly radiosensitive. The smaller neoplasms are more radiosensitive than the larger growths. A report is made on the pathologic findings of eight eyes containing retinoblastomas. The results of a clinical study of 67 eyes treated by the author and others are also presented.

The histologic findings in treated eyes may be summarized as destruction of choroidal and retinal blood vessels, disorganization and degeneration of adjacent retina, increase in size of nuclei of tumor cells due to disintegration, and coagulation necrosis of mitotic figures and cells. The author uses circular radium discs molded to fit the sclera and attached by silk sutures. The radium loading is calculated to deliver a dose of 3,500 r per week. This represents a maximum dose.

The clinical studies show that when ⅓ or less of the retina is involved radiation may effect a cure. When ½ or more of the retina is involved the results were bad. In advanced cases, enucleation of the globe with 10 mm. of nerve is advised. The orbit should be irradiated postoperatively.

G. S. Tyner.

18

SYSTEMIC DISEASE AND PARASITES

Iannaccone, A., and Kornerup, T. **Plasma lipids and diabetic retinopathy.** Acta Med. Scandinav. **148**:411-416, 1954.

One hundred diabetic in-patients were studied, forty-one of whom had diabetic retinopathy. At the time of study all had their diabetes well under control. Their blood was examined in respect to total lipids, cholesterol, lipid phosphorus, and cholesterol-lipid phosphorus ratio. The only statistically significant difference found between the diabetics with retinopathy, and those without was that the average total lipids were higher in the former group. The authors considered that this might possibly be related to increased adreno-cortical activity. (A second study showed no significant difference in plasma lipids in diabetics with atherosclerosis, and those without it.) Harry Horwich.

Landaburu, P. B., Dávalos, R. C., and Jolly, C. H. **The relationship between the severity and care of diabetes mellitus and the incidence of diabetic retinopathy.** Arch. oftal. Buenos Aires **29**:7-21, Jan. 1954.

A careful statistical analysis of 100 patients who were known to have had diabetes mellitus for no less than 10 years, leads the authors to adhere to the opinion that, all other factors being equal, retinal changes are more apt to occur in poorly treated patients than in those submitted to prompt, efficient treatment, adequate from the start and continued without intermissions. (12 tables, 7 charts)

A. Urrets-Zavalía, Jr.

Lodato, G. **The sign of Baillart in epileptics.** Gior. ital. oftal. **7**:57-63, Jan.-Feb., 1954.

A study of the tension of the retinal arteries in 42 epileptics was made. The majority showed a rise of the minimal

tension of the retinal arteries (Baillart's sign), and if this is an indication of the intracranial pressure, its presence would indicate an increased intracranial pressure in patients with epilepsy. Examinations were carried out during the quiescent periods, as examinations during crises by various authors had shown wide variations. Baillart's sign was positive in a larger proportion in those patients who were prone to convulsions. (1 table, 6 references) V. Tabone.

Long, Kathleen M. **Congenital toxoplasmosis.** Brit. J. Ophth. **38**:123-125, Feb., 1954.

A case report of congenital toxoplasmosis is presented. This is of interest because an original misdiagnosis of congenital syphilis was made many years ago. Although the dye tests on both mother and daughter are negative the daughter has X-ray findings of calcified opacities suggestive of toxoplasmosis, and the mother has a positive complement-fixation test. The latter probably indicates that the infection is still active.

Orwyn H. Ellis.

McCurry, A. L. **The Stevens-Johnson syndrome.** Tr. Ophth. Soc. U. Kingdom **73**:103-109, 1953.

The purpose of this paper is to draw attention to the fact that Stevens-Johnson syndrome is much less rare than is generally known. During six months the author saw six cases of it. The Stevens-Johnson syndrome is described as a bilateral conjunctivitis associated with a rash and constitutional symptoms of ordinary respiratory infection, or a toxic reaction. The rash consists of erythemato-papular lesions which may go on to vesiculation and bullae. There may be painful dysuria and retention. No specific drug was found to cure the disease but penicillin and aureomycin appeared to control the co-existing bacterial infection. The eye manifestation required the simplest symptomatic treatment. Beulah Cushman.

Minsky, Henry. **Numerical evaluation of ocular signs of essential hypertension.** Mt. Sinai Hosp. J. **21**:29-37, May-June, 1954.

This investigator sought to establish a classification based on unequivocal objective signs of hypertension in the eye. Topographically he divided the arterioles into branches or segments, noting that narrowing is obvious only when the ratio of the arteriole to its branch is 10:6 or less. The signs were then classified under optic nerve head, retinopathy, arteriolar narrowing, venule narrowing, and abnormality of the arterio-venous crossing. A code was devised to record the type, location, and degree of each finding. Each sign then was given a weighted value. The sum of all the signs' values after careful examination was then multiplied by a constant, which resulted in the diastolic pressure. It was believed that these calculated values for diastolic pressure were dependable for base line estimations; so much so that a calculated pressure less than the sphygmomanometric reading might indicate that the latter was raised because of psychogenic stimulation, and a calculated pressure greater than the sphygmomanometric reading might indicate that the latter was misleadingly low because of cardiac dysfunction.

Harry Horwich.

Pineroy y Carrion, Antonio. **Associated oculo-cutaneous diseases.** Arch. Soc. oftal. hispano-am. **13**:1287-1312, Nov., 1953.

The literature is reviewed. (33 references) Ray K. Daily.

Sapuppo, Concetto. **Syndrome of Morgagni with an angioma of the retina.** Gior. ital. oftal. **7**:44-56, Jan.-Feb., 1954.

Sapuppo describes a patient with the syndrome of Morgagni (endocraniosis with frontal hyperostosis, obesity, hirsut-

ism, arterial hypertension and alterations of the cerebral venous system) and an angioma of the retina, close to the disc. The various possible causes of association of ocular and cerebral vascular abnormalities are discussed, and the hypothesis is advanced that, in this case, the venous stasis may have caused the changes which constitute the syndrome of Morgagni. (4 figures, 29 references) V. Tabone.

Sybar, Kenneth C. **Endocrine exophthalmos.** Tr. Ophth. Soc. U. Kingdom **73**:639-650, 1953.

The literature on endocrine exophthalmos is reviewed. The author raises the question of accepting any hypothesis based on the conception that thyrotropic hormone is directly responsible for the production of progressive exophthalmos following thyroidectomy, since thyrotoxicosis is relatively infrequent and primary myxedema is seldom, if ever, associated with exophthalmos. Furthermore, nontoxic goiter is never associated with the development of exophthalmos despite the fact that there may be some increase in the level of thyrotropic hormone in the blood and an excessive amount of thyrotropic hormone in the blood in cases of progressive exophthalmos is not a constant finding. Research has been extended to include the effect of androgens which cause an increase in exophthalmos and a possible link between adrenocorticotropic hormone and the production of exophthalmos.

All possible measures must be taken to ensure the integrity of the eyeball during the active phase of the proptosis. Thiouracil therapy probably carries less risk for the eye than thyroidectomy in such cases.

Beulah Cushman.

19

CONGENITAL DEFORMITIES, HEREDITY

De Bernardinis, E. **Cataract associated with skin lesions.** Gior. ital. oftal. **7**:3-10, Jan.-Feb., 1954.

The association of cataract and skin lesions is reviewed and a case with Werner's syndrome is described in detail. The patient is a woman, 35 years of age, with sclerodermal lesions of the skin, disturbances of phonation, bilateral cataract of the juvenile type, and fronto-basilar endocraniosis. (6 figures, 8 references)

V. Tabone.

De Oliveira, Luiz V. **Ectopia lentis.** Arq. brasil. de oftal. **16**:195-202, 1953.

Three patients with bilateral ectopia lentis were observed, all of whom had defective vision after correction. Since the patients were related, the hereditary character of the anomaly was investigated as well as possible. Three instances were discovered in four generations of the affected family, whose genealogy is presented for study. The author discusses heredity and the mode of transmission of determining genes. Hereditary eye diseases are mentioned to illustrate the importance of genetics in relation to ophthalmology. Ectopia lentis is considered an hereditary disease, transmitted as a dominant type. Its limited appearance is explained by poor penetration of the responsible gene. The condition may also occur as a sporadic anomaly, according to Ida Mann. (5 references) James W. Brennan.

20

HYGIENE, SOCIOLOGY, EDUCATION, AND HISTORY

Ainslie, Derek. **Ophthalmic nursing.** Tr. Ophth. Soc. U. Kingdom **73**:565-569, 1953.

In the training of an ophthalmic nurse the practical side is given precedence and a nurse well trained in the general aspects of nursing is essential to the eye physician. Any ophthalmologic training should be entirely complimentary to the general training. Beulah Cushman.

Cross, A. G. **Individuality in ophthalmology.** Tr. Ophth. Soc. U. Kingdom **73**: 49-54. 1953.

The objective in the education of ophthalmologists is to make them capable of diagnosing all ocular abnormalities and to apply the necessary treatment, modified to suit the requirements of the particular patient. A comprehensive knowledge of ophthalmology should be based on adequate foundations of physiology and pathology. It is suggested that each one aim to be an expert of experts in some small part of our subject. It is important to remember the psychologic aspects of ophthalmic disease. The young student should assist, watch and carry out some operative procedures, he should visit other clinics, carry on some research problems under direction, and also take time to improve his knowledge of his own language.

Beulah Cushman.

Duke-Elder, S., Neele, J. C., and Riddell, W. J. B. **Symposium. Visual requirements in relation to modern travel.** Tr. Ophth. Soc. U. Kingdom **73**:287-347, 1953.

Duke-Elder, Sir Stewart. pp. 287-296.

The lowered visual requirements for the army and drivers of public vehicles have been inspired by the lessening of prejudice to the use of spectacles and their protection of drivers, the realization of the complete inadequacy of the present visual standards, and, as the speed and density of traffic increase, the relatively small part played by vision and the immensely more important part played by the driver's psychologic make-up.

Visual standards of today require a measurement of visual acuity taken in daylight, sometimes color vision, and occasionally visual fields. For visual acuity the author recommends Landolt's rings in which speed and accuracy are factors. Isochromatic tables are probably the most useful in color vision and the field of vision can be checked by the confrontation test. Evidence seems to suggest that the mind plays a much more important part in the causation of accidents than reduced vision.

Tests for drivers should be psychophysical tests, such as reaction time, resistance to distraction, vigilance, eye and hand co-ordination, judgment of spatial relationships and speeds, and a performance test of driving in a stationary car while a projector throws a moving landscape on a screen. The efficient traveler of the future will probably require no visual standards at all but will rely solely on electronic means of navigation.

Neeley, J. C. pp. 297-331.

Visual capacity may affect air travel and air travel may have its effect on visual capacity. Modern air travel is conducted more and more by instrumental vision. Visual warning systems were set up but subsequent investigation revealed that good vision is of prime importance in learning to fly and that subjects with horizontal heterophoria over 6 p.d. were not acceptable. A qualifying standard for stereoscopic vision was not found to be satisfactory and those considered to have defective color vision in practice have been found to have a color sense sufficient for aviation. Pressure suits devised to protect the aviator against physiological onslaught may soon be as anachronistic as the suit of armor made in 1575 to protect the warrior from the physical onslaught prevalent at that time. These and innumerable other details are discussed at length.

Riddell, W. J. B. pp. 333-347.

Vision is essential even though the movement of vehicles has increased from equestrian to supersonic speed in a short lifetime. Signaling systems aid by employing both auditory and visual signals such as colored flags, lights, and various shapes. Rear lighting of motor vehicles with a minimum of 0.25 candela has become necessary. The candela is such that the luminance of a black body or Planck-

ian radiator at the freezing point of platinum is 60 candelas per square cm. Yellow fog lights have not been adequate. Peripheral vision causes illusions which contribute to safety, as an object exposed momentarily in the periphery appears to be nearer to fixation than is the case. The dazzle and glare problem of the road has not been solved satisfactorily.

Beulah Cushman.

Hansell, Peter. **Illustrating the lecture.** Tr. Ophth. Soc. U. Kingdom **73**:363-371, 1953.

The author gives a wealth of advice on the use of projected illustrations in different forms. When text is used it is well to confine the material to an amount which can be put on a postcard. View the copy at a distance eight times the greater dimension of the card, if you can not read it, neither can the audience. Tables should be reduced to graphic form. Miniature slides are cheapest and lightest and make a most flexible form of lecture material, as there is economy of space, weight and materials, and the size is an international standard. Color is equally available at little expense. It is possible for the lecturer to have his own miniature projector and collection of slides and they can be easily carried in a car. Short lengths of ciné film deserve a special place. Stress must also be placed on the understanding of the projector and the instruction to the projectionist. Beulah Cushman.

Lopez, Silvan. **The ophthalmologist and public health.** Arch. Soc. oftal. hispanoam. **13**:1345-1376, Nov., 1953.

The author believes that an ophthalmologist should direct the campaign against trachoma, leprosy, ophthalmia neonatorum and epidemic kerato-conjunctivitis; should supervise the treatment of contagious ocular diseases, the detection and treatment of retrolental fibroplasia, the treatment of all cases of strabismus from the moment they are detected, the systematic ocular examination of all children five years of age, the control of ocular infections in the schools, and a complete ocular examination at the end of the scholastic period. An ophthalmologist should participate in developing a program of school hygiene, vocational guidance, supervision of ocular hygiene in industry, and prevention of industrial ocular accidents. The same services should be supervised by an ophthalmologist in the Public Health and military services. The author points out that the scientific advances have widened the fields in which the ophthalmologist can serve the individual and the community. (15 references) Ray K. Daily.

MacKellar, M. B. **Hospital facilities and nursing procedures.** Tr. Ophth. Soc. U. Kingdom **73**:569-584, 1953.

The eye hospital facilities vary according to the geographical position, the fabric of the hospital, the personnel, and the quality of the staff employed. Separate wards are wanted for patients with cataract, retinal detachment, squint, and glaucoma; others for those with infectious lesions and inflammatory conditions. An isolation room is desirable in an eye hospital. The visual environment should include restful color, dimming lights, and dark blinds for ophthalmoscopic examination. The stairways should have adequate guards. Auxiliaries should be trained as assistant nurses, and they can play a very important part in ophthalmic nursing.

Beulah Cushman.

NEWS ITEMS

Edited by Donald J. Lyle, M.D.
601 Union Trust Building, Cincinnati 2

News items should reach the editor by the 12th of the month but, to receive adequate publicity, notices of postgraduate courses, meetings, and so forth should be received at least three months before the date of occurrence.

Deaths

Dr. Ossian Homer King, Hot Springs National Park, Arkansas, died June 15, 1954, aged 67 years.

Dr. Hugo Bruno Charles Riemer, Norwood, Massachusetts, died July 10, 1954, aged 78 years.

Announcements

Military surgeons convention

The 61st annual convention of the Association of Military Surgeons of the United States will be held November 29 and 30 and December 1, 1954, at the Hotel Statler, Washington, D.C. The program will cover a wide range of professional and scientific subjects. As in the past, one of the outstanding features will be the honors-night dinner on December 1st, at which the Sir Henry Wellcome Medal and Prize, the Gorgas Medal, the Stitt Award, the McLester Award, the Louis Livingston Seaman Prize, and the Founder's Medal will be presented.

Georgia meeting

The annual meeting of the Georgia Society of Ophthalmology and Otolaryngology will be March 11 and 12, 1955, at the General Oglethorpe Hotel, Savannah, Georgia. The speakers will be: Dr. Francis Heed Adler, Philadelphia; Dr. J. W. McCall, Cleveland; Dr. J. A. Hilger, Saint Paul; Dr. Walter H. Fink, Minneapolis; Dr. James H. Allen, New Orleans; and Dr. P. E. Ireland, Toronto.

Walter Reed postgraduate course

A short postgraduate course in ophthalmology will be held at Walter Reed Army Medical Center on February 2, 3, and 4, 1955. The following civilian guest lecturers are supplemented by military ophthalmologists on the program: Prof. Marc Amsler, Zurich, Switzerland; Dr. J. S. Friedenwald, Dr. W. F. Hughes, Jr., Dr. P. C. Kronfeld, and Dr. L. E. Zimmerman.

The meeting is open to military and civilian ophthalmologists with no fee. Applications for military personnel should be processed through channels in accordance with SGO Circular No. 43, dated 18 May 1954. Civilians who wish to attend should apply to the Commanding General, Walter Reed Army Medical Center, Washington 12, D.C., Attention: Colonel J. H. King, Jr., Course Director in Ophthalmology.

All-India meeting

The All-India Ophthalmologic Society will hold its annual meeting from February 16 to 19, 1955, in Bombay, India.

In behalf of Dr. S. N. Cooper, secretary of the society, a cordial invitation is extended to United States ophthalmologists and their families to attend the meeting and to take part in the scientific phase of this conference. There will be a symposium on "Diseases of the lacrimal apparatus." However, papers on other ophthalmologic subjects may also be read.

The Ansett Travel Service, 323 Geary Street, San Francisco, is in charge of all travel arrangements. The tour will originate in San Francisco, take six weeks and will include stopovers in Honolulu, Tokyo, Hong Kong, Bangkok, the Taj Mahal, New Delhi, Bombay, Cairo, Rome, and New York.

The all-inclusive costs including first-class air transportation, meals, hotels, and excursions will be $2,800.00 per person.

Association for research—Southern Section

Dr. A. E. Meisenbach, Jr., secretary of the Southern Section of the Association for Research in Ophthalmology, announces the following program to be held in conjunction with the Southern Medical Association meeting at Saint Louis, Missouri, November 9, 1954. The scientific session will be held in private dining room 7 of the Jefferson Hotel at 6:30 p.m. Members and guests are cordially invited to attend the program:

"Adenocarcinoma of Harder's gland in mice," Dr. Walter H. Benedict, Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee; "Some biochemical characteristics of hydrochloric-acid burns of the cornea," Marion A. Guidry, Ph.D., Department of Ophthalmology, Tulane University of Louisiana, New Orleans; "The cultivation of ocular tissues," Dr. Robert Hoagland, Department of Ophthalmology, Tulane University of Louisiana, New Orleans; "The role of neural reflexes in anaphylactic type reactions in the rabbit eye," John Schmedtje, Ph.D., Instructor, Anatomy Department, Saint Louis University School of Medicine, Saint Louis; "Experimental transplantation of heterologous and lyophilized cornea: A preliminary report," Dr. Louis Daily, Jr., and Dr. Stuart A. Wallace, Departments of Pathology and Ophthalmology, Baylor University College of Medicine, Houston, Texas; "Light pattern test of visual field," Dr. Albert N. Lemoine, Jr., Kansas City, Missouri.

Pennsylvania clinical meeting

The fifth annual midwinter clinical meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held at the Eye and Ear Hospital, Pittsburgh, on Wednesday, January 19, 1955. The one-day meeting will consist of operative clinics and symposia. At this meeting final arrangements will be made for the May 18 to 21, 1955, meeting at the Hotel Traymore, Atlantic City, New Jersey.

New Orleans midwinter convention

The midwinter convention of the New Orleans Academy of Ophthalmology, to be held January 31 through February 4, 1955, will feature a symposium on ocular motility at which the principal speakers will be: Dr. Francis Heed Adler, Dr. Harold W. Brown, Dr. Hermann M. Burian, Dr. Frank D. Costenbader, Dr. Walter H. Fink, Dr. George P. Guibor, Dr. Philip Knapp, and Dr. Kenneth C. Swan. The program will also include round-table discussions. The registration fee of $75.00 includes associate membership in the academy for the year 1955, as well as all other features of the convention. Hotel reservations should be made early by writing directly to the Jung Hotel, 1500 Canal Street, or to the New Orleans Academy of Ophthalmology, 211 South Saratoga Street, New Orleans.

Gill spring congress

The Gill Memorial Eye, Ear, and Throat Hospital, Roanoke, Virginia, will hold its 28th annual spring congress on ophthalmology and otolaryngology, April 4 to 9, 1955. Among the guest speakers will be:

Dr. Henry L. Birge, Hartford, Connecticut; Dr. Paul Boeder, Southbridge, Massachusetts; Dr. William B. Clark, New Orleans; Dr. Dan M. Gordon, New York; Dr. Anderson C. Hilding, Duluth, Minnesota; Dr. Chevalier L. Jackson, Philadelphia; Dr. Bertha A. Klien, Chicago; Dr. T. G. Martens, Rochester, Minnesota; Dr. Daniel S. Miller, Boston; Maj. Gen. Daniel Ogle, Washington, D.C.; Dr. Thomas Paine, Ann Arbor; Dr. R. Townley Paton, New York; Dr. James Purnell, New York; Dr. A. D. Ruedemann, Detroit; Dr. Robert E. Ryan, Saint Louis; Dr. Richard Schneider, Ann Arbor; Dr. John Sheldon, Ann Arbor; Dr. Grant Ward, Baltimore.

Research Study Club

The Research Study Club of Los Angeles announces its 24th annual midwinter clinical convention in ophthalmology and otolaryngology, January 17 through January 28, 1955. Instruction courses in ophthalmology will be given by Mrs. Helenor Foerster and Dr. Robert H. Shaffer of San Francisco. Among the guest lecturers for the eye program will be Dr. Edmund B. Spaeth and Dr. Irving H. Leopold of Philadelphia. For further information write to: Dr. Pierre Violé, 1930 Wilshire Boulevard, Los Angeles 57, California.

Miscellaneous

Proctor Laboratories dedication

At the dedication of the Francis I. Proctor Laboratories, University of California School of Medicine, San Francisco, September 29th and 30th and October 1st, the following papers were presented:

"Research in ophthalmology," Dr. Alan C. Woods, Baltimore; "The beginning and the early years of the pathology laboratory at the Massachusetts Eye and Ear Infirmary," Dr. Frederick H. Verhoeff, Boston; "Research in ophthalmology in England," Sir Stewart Duke-Elder, London; "Heterochromic cyclitis (Fuchs' syndrome)," Dr. A. Franceschetti, Geneva, Switzerland; "Carbonic anhydrase inhibition and aqueous flow," Dr. Jonas S. Friedenwald, Baltimore; "Studies on the canal of Schlemm," Dr. Georgiana Dvorak-Theobald, Chicago; "Ophthalmia neonatorum," Dr. Hugh L. Ormsby, Toronto; "Precancerous and cancerous melanosis of the conjunctiva," Dr. Algernon B. Reese, New York; "Studies on the vitreous," Dr. S. Rodman Irvine, Los Angeles; "Responses of intraocular and blood pressure to electric stimulation of the diencephalon: The ninth Francis I. Proctor Lecture on Ophthalmology," Dr. Ludwig von Sallmann, New York.

"Surgery of the dislocated lens," Dr. Derrick Vail, Chicago; "Some recent contributions to the problem of trachoma," Dr. Giambattista Bietti, Parma, Italy; "Congenital entropion and hypotropia: A clinical entity," Dr. Kenneth C. Swan, Portland, Oregon; "Fundus changes in hypertension in relation to postmortem systemic alterations," Dr. M. Puig Solanes, Mexico City, Mexico; "Tonographic studies of the glaucoma secondary to delayed restoration of the anterior chamber," Dr. Peter C. Kronfeld, Chicago; "Retrolental fibroplasia," Dr. Norman Ashton, London; "Further investigations on the anatomy of the origin of the ophthalmic artery," Dr. Josef Böck and Dr. H. Schwarz-Karsten, Graz, Austria; "Conjunctival implants in the rabbit anterior chamber," Dr. David G. Cogan, Boston; "Nutritional amblyopia," Col. John H. King, Jr., Washington, D.C.

Personal

At a meeting of the Israel Ophthalmological Society held at Hebrew University, Jerusalem, August 1st and 2nd, Dr. Isadore Givner, New York, presented a paper on "Ocular expressions of systemic diseases."

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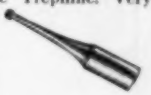

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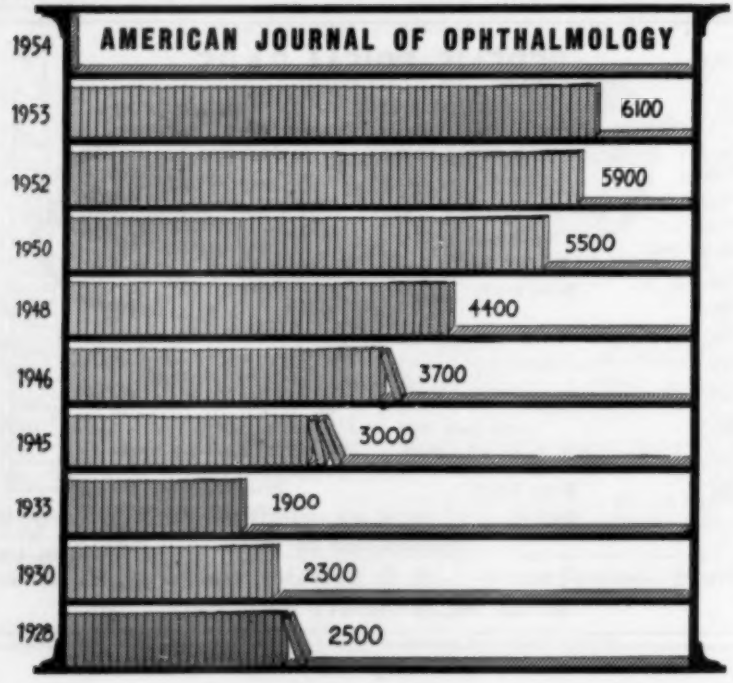

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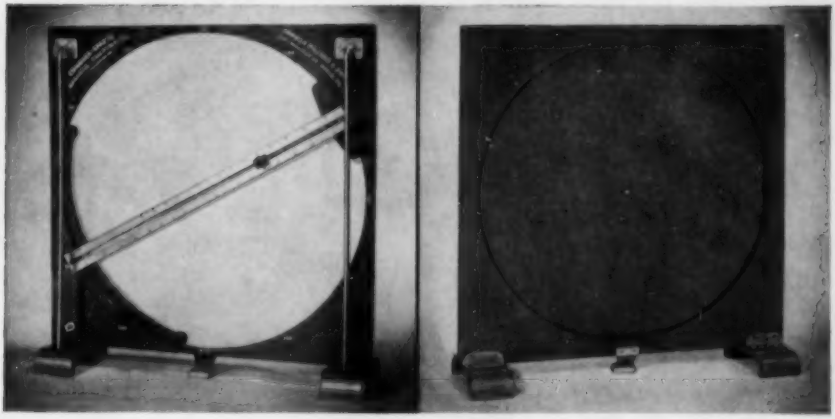

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